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CIVILIZATION'S FOOD AS THE POSSIBLE ORIGIN OF DENTAL DISEASE.[1]

By THOMAS T. ALKIN,
Sydney.

I UNDERSTAND that by mutual agreement the problem of systemic troubles from dead teeth and apical areas of infection is not under consideration this evening, having been well discussed at a previous meeting. In any case, the serious results, together with treatment and elimination, are definitely established. The subject now for review is the possibility of dietetic origin in periodontal disease and caries. It is of the greatest interest to those of us who can go back in dentistry to the time when there were almost as many theories as to the starting point of these unfortunate dental conditions as there were interested dentists; and these theories were as widely opposed as the poles. With each passing year numbers of these faded out and gradually more concentrated study gave place to wild guessing, so that, looking back, we can follow the lines of thought converging, till today they are narrowing down to a probability of a definite focus upon the true origin. As the position is now, in this smaller area, we find the question of dietetics occupying the major part of the field. In times past this possibility was rarely suggested, whereas today it fills the greater portion of dental literature. True it is that wide differences of opinion exist as to what is wrong with civilization's food and food manipulation, but as all the argument deals with classes of food, preparation, loss of minerals and vitamins, soil deficiency and such, it is not removed from the classification of dietetics; and possibly the warmth of argument is the melting

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and of the New South Wales Branch of the Australian Dental Association on August 30, 1934.

pot from which something of value to humanity will eventuate in the prevention of dental diseases. Dentists who wish to be something more than all-time repairers of teeth, feel their responsibility keenly, and the difficulty in seeing light through the mass of conflicting opinions, owing to the many side-tracks confusing the issue (as when dietetics is interwoven with the soils producing food), renders it necessary that research must combine medical, dental and possibly agricultural experts taking part in the work. From our research workers, both medical and dental, collecting evidence in all parts of the world, among all nations and primitive races, the proof showing some serious and damaging alteration in our foods is becoming overwhelming. Therefore it seems likely, given helpful and friendly cooperation between medical men and dentists, that, hidden in the various treatments, handling, manufacture, denaturing and demineralizing of natural food products (or even in the soils growing them, to which I will refer later on), we shall locate the cause. If we are successful in this, and it is shown that the mistakes are of our own creation, it is logical to assume that the remedy also will be within our control.

It is not my intention, nor is it possible to do more than try to place before you, from the enormous amount of writings on this subject, some of the findings of the various men doing this research, hoping that a discussion with the medical profession will help us to weigh the evidence better and allow us to look at it from angles from which their greater knowledge views it. There is no need to enlarge upon the ravages of dental diseases, but merely to give a bald statement or two of facts as to their extent today. It is generally agreed, as shown by authentic statistics, that over 86% of civilized peoples, and also the more primitive races, after certain periods of contact with our food habits, are suffering from caries, or lowered resistance in mouth tissue, with periodontal disease in marked evidence or predisposition to the same. Our dental history tells us that up to the middle of the seventeenth century the mouth conditions of the people of the British Isles were splendid, but recent statistics show an appalling state of deterioration, resulting, as the consensus of opinion indicates, from a diet lacking in the mineral substances or from some want of function upon which bones, teeth and tissue depend (and, no doubt, from a vitamin deficiency as well). Half the children of one school in Leeds were found by Dr. Hull to be suffering from mineral starvation, and American reports show similar findings. *The British Medical Journal* (I think it was of August, 1920) had an article on this subject and drew attention to the fact that before the war only 5% of children were free from active caries, whereas after the war 44·4% were free; and it was also stated that the war-time diet of harder foods, stale bread of less refined flour, the eating of all crusts, the rationing of sugar and luxuries, seemed to be the only logical possible reasons for the remarkable improvement. Dr. Weston Price has concluded extensive investigations in many parts of the world, and from his voluminous writings I select the following points. He noted particularly in parts of Switzerland, where isolation and difficulties of transport forced the people to depend entirely upon their own produce for food, that the teeth were in splendid condition; but in tracing up members of families who had left home some years before and had taken up residence in the larger cities, he found their teeth and mouths seriously broken down as compared with those of their relatives in the more inaccessible mountain valleys. His research in other parts gave the same findings.

It is common knowledge among American dentists, in confirmation of the above, that young immigrants arriving from the southern States of Europe have splendid teeth, but that these teeth deteriorate rapidly after a period of residence in the United States. Medical men report similar findings among the isolated Arabs of the Sahara, where diseases of the teeth are said to be almost unknown. As far back as 1795, Weld, writing of American-born men and women of that time, states: "They had decayed teeth at an early age." He thought that this was due to the general use of salted provisions. He eliminated climate as a causative agent, since the Indians had perfect teeth. In 1861, Catlin, speaking of the Sioux Indians, said:

> I was forcibly struck with the exceptional completeness and soundness and total absence of malformation of their beautiful sets of teeth of all ages. The teeth arrange themselves regularly as keys of a piano, their teeth do not decay, but preserve their soundness and enamel, with powers of mastication, to old age.

McCollum sums up on "diet in relation to teeth":

> It is evident that certain peoples in the past and certain people of the present time have escaped the ravages of dental disease, which is now such a heavy burden in America and certain European countries. The evidence points to the failure to form teeth during the developmental period which have within themselves adequate barriers of defence against the agencies from without which would destroy them, as the chief cause of tooth decay. The development of sound teeth capable of resisting the destructive agencies mentioned is essentially a dietary problem. The small jaws and crowding of teeth so frequently seen is almost certainly the result of faulty skeletal development and will be influenced by the dietary errors which favour development of rickets.

Dr. Campbell writes:

> I have again and again referred to the prosaic fact that there are among the inhabitants of this country some 200,000,000 carious teeth, as many alveolar abscesses (*pyorrhœa alveolaris*), and some 30,000,000 root abscesses, for the most part preventible by the simplest possible means. It is impossible to exaggerate the state of the teeth in this country; it constitutes a national disgrace. Dr. Sim Wallace has, time after time, debated this matter with me, and we have long felt that the remedy can only come through the medical profession. Of the problems we have to face I put the food problem first, convinced as I am that it is the most urgent and that more preventible disease is due to faulty eating and drinking than any other cause.

Sim Wallace, M.D., referred to in the last quotation, in *The Medical Press*, London, writes:

> Pyorrhœa also is almost always of dietetic origin.

Then he deals with the effect of food, ending up by saying:

> These foods will not only prevent the disease but, even after it has started will arrest it, and distinguished dentists have noted the good effect of these foods in its treatment. Indeed, until this was recognized, treatment was invariably unsatisfactory.

B. Donaldson, M.D., speaking of scurvy and pyorrhœa, says:

> Scurvy may effect tooth conditions by the teeth actually dropping out.

He concludes:

> It is worth noting that scurvy was completely absent where native diets are used. There is no racial immunity to scurvy, which appeared first when Esquimaux were employed by white men and got scurvy along with their masters, and later, when the increased price of furs induced Esquimaux to buy groceries for skins instead of flesh foods by direct hunting.

I may enlarge upon Dr. Donaldson's remarks, for these Eskimos, as we find from our research, have admittedly the best teeth of known humans (the old time Maoris possibly had equally good teeth), where they are isolated from contact with traders and others; and Stephanson, in his book on the Arctic, says that they have no word in their language to mean toothache, but where these people have constant contact with Americans, Canadians and others, with access to their foods, the mouth conditions are notably bad. We have this also forcibly brought home to us in our own Australian blacks, before and after their association with our foods and drinks. In the free dental clinics of Virginia it is noted that a much smaller percentage of negroes are treated. The negro child eats, as soon as it can, solid food needing use of masticating power, with quantities of greens, cabbage, turnips, salads and other vegetables; they do not have sugars and the fancy foods available to the white child, so the negro diet contains a large percentage of lime, iron and other salts, also vitamins. Supporting this, I found evidence right at our door, when recently I took part in an examination, made at the request of the then Minister for Health, of the Seventh Day Adventists at their annual camp. I found a high degree of disease resistance in the gum tissue and a wonderful freedom from periodontal disease. In no other similar number of people in this country would it be possible to find conditions anything like the same, their diet being high in mineral and vitamin content; so, if we accept the dietetic origin, this may account for the difference.

Dr. Arnold Sack, of Heidelberg, after the examination of some 30,000 mummies, wrote:

> In examination of skulls of Egyptians prior to the time of the pyramid building, it was noted that the wealthy classes on a more luxury diet had dental diseases, while the workers or slaves were free, their diet being hard dark bread, natural products with vegetables.

Stephanson sent many skulls of early inhabitants of Iceland to an American university for examination and it was found that these ancient people of about the thirteenth century were free of all dental diseases, but investigation within the past forty years reveals a steady increase of dental breakdown, the times corresponding with the importation of foodstuff and change in character of the diet. The imports were cereals, sugar, hams, sausages, sardines and the like. The dental conditions were splendid and caries was practically unknown till after this trade contact.

Dr. Colyer, of London, says of pyorrhœa:

> The prevalence of the disease is probably due to the character of the diet of the present day.

And in his investigations into the disease in Africa he makes this note:

> Wild animals when brought under domestication suffer, though in a state of nature such a thing is rare.

He believes that the ætiologies of caries and pyorrhœa are closely related and that the epoch of cooking was the one to which their modern types could be traced.

Professor Howe, experimenting on animals, mostly monkeys, with different diets, writes:

> On the normal and balanced diet, which by experience was found suitable for these animals, dental decay never developed, but when we deviated from the normal, caries of the teeth developed, varying in rapidity and extent according to the difference made in the diet.

Dr. Howe also found that by stopping the supply of vitamins and calcium from the diet of animals pyorrhœa could be induced, and cured by a return to the correct dietary. A veterinary surgeon in England drew attention to the extraordinary prevalence of pyorrhœa in pet dogs, pampered and unnaturally fed, and how quickly a cure was effected by returning them to a normal diet. In this country it is fairly common to see dairy-fed cattle wandering around eating old bones, but when moved on to lucerne or fresh pastures, with many bones available, they will not touch them. Instinct in this case points out the way to replace some craving for mineral food. Dr. Weston Price has proved that the milk of such dairy-fed cattle is greatly deficient in very necessary properties, and it is well known in Canada that the winter hand-fed cows produce milk much inferior to that of the spring, after pasturing on the young grass. When we as dentists find our literature full of such matter, is it not logical for us to assume that alterations in the food of human beings will similarly reduce the resistance of the tissue and allow the invasion of germ life as we see it in periodontal disease? It is admitted that an iodine deficiency is the cause of goitre in countries where the soil and produce has not this element, and we know that medical science recognizes a certain vitamin deficiency in scurvy and treats it by dietetic methods; so is it not more than just to assume that this lowered resistance in gum tissue has similar deficiency origin? When I was investigating these matters for the Government on the first occasion, I found in Chicago that out of ten thousand animals killed in the yards it was rare to find a diseased beast coming from natural food conditions, yet when we hand-feed and domesticate them it becomes necessary to

have a school of veterinary science to deal with the problems of animal disease, including dental attention. Some are inclined to discount the value of findings in experimental work with animals; so it may be well here to quote from the *Dental Journal of Research*, which contains notes on an interesting medico-dental experiment carried out at Hawaii.

> Although that country is subtropical and abounds in sunshine, dental decay is very common and the teeth of many babies are ravaged by decay before complete eruption. Vitamin deficiency diseases, except beri-beri, are rare; the diet of the labouring population consists of rice, small amounts of fish, bread, meat, eggs, fruits and vegetables. For the experiment a sugar plantation, with fixed population of about 4,500 Japanese and Filipino labourers and families, was chosen, and attempts were made to induce these to eat taro and potatoes instead of rice and bread, and to take more milk, fruits and vegetables. This was done through hospitals, baby clinics, antenatal centres, school meals, and propaganda. The result has been a drop in the infant mortality from 160 in 1929 to 25 in 1932. Babies fed on health centre diets are larger, stronger and have markedly better teeth.

Dr. Larsen and Dr. Pritchard, who are responsible for the experiment, express the view that under Hawaiian conditions dental caries can be prevented by preponderance in the diet of foods that yield alkaline ash—taro, potatoes, fruits and vegetables. I have been able to secure similar reports from other centres, but time will not permit me to include them here.

From my own experience, confirmed by what I learned in the various research departments in the world, I can come to no other conclusion but that periodontal disease is definitely a disease following upon lowered resistance, which allows germ life in the mouth to take advantage of such weakened conditions, there to increase rapidly; and that, in people with high resistance from correct foods, pyorrhœa is impossible. As it cannot be transmitted from one person to another, therefore the patient has to provide the conditions by this systemic weakness. Trauma and many other things classed as causes must become secondary again, because vast numbers of mouths with striking irregularities of articulation are disease-free; also it is impossible to believe that all teeth of the immune races have perfect articulation. (If we were to admit trauma as the origin, then of necessity numbers of people whose teeth do not make perfect contact, in countries where the disease is unknown, should have it, for it cannot be that this irregularity is never present.)

We have definite information from New Guinea that serious bacteriological invasion of the supporting tissue round the teeth, and classed under the name of New Guinea mouth, responds rapidly to a diet of oranges and yeast extract, much in the same way as a return to normal is obtained in scurvy on a high antiscorbutic diet.

West China Union University and the University of Toronto report interesting experiments conducted on animals, where it has been possible to produce or inhibit caries by regulating the phosphorus and vitamin *D* content of the diet; also other investigators, whose reputations stand for something in medicine and dentistry, have instanced cases in which, by dieting alone, extensive caries has been arrested and calcification has taken place over almost exposed pulps in children's teeth. Incidentally they report improved mentality. Without having the time, means or ability for research along these lines, I have to take a common sense view of the work of such earnest research students; and it is impossible to get away from the sincere belief that caries and periodontal disease are directly associated with habits and customs. These habits and customs have been introduced as we have deviated from a more natural dietary to the present state, where we have a far greater luxury diet with highly refined and machine preparation of vital parts of our staple foods, sacrificing more of the natural and necessary for the refined, manufactured and demineralized. The early breakdown of the immune races after association with our foods must eliminate all other possibilities; the time factor and impossibility of other customs of life being introduced must confirm this. I find in my reading upon this subject that after every famine due to the failure of a staple article of food, such as the potato crop in Ireland, America, Norway and other places, scurvy has broken out, with the usual mouth symptoms in evidence.

To the medical section of this meeting I should like to say how much we appreciate this opportunity of discussing the things that are agitating our minds and the position we should take up in relation to these problems, and I feel sure that some of the points I shall raise in concluding this paper will be answered in a way which will materially assist us.

If the dietetic origin of caries and periodontal disease is admitted, our difficulties are magnified and the whole situation of this portion of dental treatment changes. If these diseases start in lowered resistance from systemic disturbance caused by food errors extending over long periods, the dentist comes into the field to treat only a symptom of another condition. Without a clear understanding of food values and cooperation with medical men in raising the resistance, our efforts can at the best give only temporary relief, and in the case of caries must mean a lifelong repair service. The prenatal diet of the mother becomes an important factor, for a deficiency in her diet lays the foundation of dental troubles which come to us in the child many years after the original damage has been done. We know that months prior to birth the teeth of the child are in preparation, and we do not see the prospective mother at this time to make any suggestion as to the necessary foods to build strong disease-resisting teeth, so when we hear the teeth blamed for numbers of systemic troubles it would be unfair to place responsibility upon the dentist. Hippocrates said that diseases should be combated at their origin, but if we admit that periodontal disease comes as a link in a chain of events starting years back in dietetic errors, the

dentist's work is far removed from the great early physician's outlook.

Another important viewpoint of interest both to medical men and dentists arises in that we can see the condition of mouth tissue and teeth in pyorrhœa, but we cannot see the internal organs and membrane. Is it not therefore most likely that some changes are taking place out of sight, arising from the original cause? And if so, does extraction of teeth without other treatment do more than get rid of a visible symptom? I ask this because in very many cases after extraction in advanced pyorrhœa the absorption of the process and bones continues and often to such an extent that wearing artificial dentures with hope of proper usefulness is impossible. When deficiency is admitted, preparations are available in highly concentrated form to attempt to replace by this means calcium, phosphorus *et cetera*; and it is a matter of great interest to dentists to determine the amount of these necessary substances absorbed by the system when they are administered in such concentrated form, compared with the amount absorbed from foods rich in these minerals. Also, in early adult life and after, what possibility is there that such preparation will reach the far-removed broken-down teeth and tissue in that area with beneficial results?

We know that in scurvy and New Guinea mouth the tissue responds to correct diet, and that being so, is it not fair to assume that in pyorrhœa similar treatment is indicated? When there is direct evidence that the mother's diet was deficient, the teeth of the children are often correctly or even beautifully formed, but examination of most minute openings through enamel, when opened up, shows that proper calcification has taken place only on the outer walls. When this occurs, is it possible that any diet, however high in mineral content, will rectify the prenatal deficiency? We are faced with many side-tracks when trying to locate the wrong in our food supplies, and I referred earlier to the possible part soils play in reducing the value of some of our staple foods. I mention this for the reason that it continually appears in the writings of research workers; and in a report I received lately from Hawaii the Agricultural Experimental Station states that tests of virgin upland soil gave 4·59% of calcium, but after one cropping it was reduced to 2%, or by more than half. If these findings are general in all agricultural areas, may it not have the same bearing upon teeth, bones and tissue that the admitted iodine deficiency in soils has upon goitre? If consideration is given to this aspect of the question, then, when fertilization is undertaken, health experts should have a lot to say as to what replacements must be made in the farming lands. It is likely that under the present system more attention is given to bulk and properties for milling and commercial purposes and that the other has escaped attention.

I have an uncomfortable feeling that I have mentioned nothing that is not better known to the medical men present, and I may not have made the best selections from the writings available to me, but I feel sure you will realize the difficulties in preparing a paper for such a gathering. When one finds a high English medical authority publicly stating that at least 20% of all chronic diseases in that country are due directly or indirectly to the teeth, and Sir Rickman Godbee writing that he has traced up over twenty-eight systemic diseases as arising from the oral cavity, it surely is time that opportunities were given for greater interchange of ideas on these subjects between the professions. I should like to go further than this and suggest that in the event of serious consideration being given to the dietetic origin of caries and periodontal diseases, a combined committee, selected from interested practitioners of both associations, take the matter up for further investigation. There are many points I should have been glad to raise tonight, but time will not permit. I mention two. One is the excessive amount of refined sugar consumed, and the other is the part certain glands may play in controlling the functioning of minerals when they are in sufficient quantities in the daily food.

In conclusion, may I say that years ago Dr. Hunter, of London, forcibly brought the dental profession to consideration of certain conservative methods of practice which at the time were taught as right and proper, resulting in a definite understanding of this side of focal infection, with wonderful results to the health of the public. When we face and settle the question under discussion we shall be a long way advanced in the prevention of our dental troubles.

If we are to benefit by study as to what is good in the diet of the immune and what is wrong in our own, we shall have to start soon, as these primitive races are fast altering their natural food habits. To one who is not a trained research worker this seems the logical starting point. When contact is made with these isolated peoples who, like the Eskimos, have small variety in their own foods, new foods introduced must of necessity be few also, so in what these people displace of their own and use of ours the injurious factor should be more easily located as a basis from which to extend investigations. There is authentic evidence that even in the natural foods of some black races very slight deviation swings the balance from good health to disease, and a quick return to health takes place when the normal food balance is restored.

I have tried to state a case from the dental viewpoint, and if the dietetic origin is established, Dr. Charles Mayo, who years ago threw out a challenge that "the next great step in preventive medicine must come from the dentists", will have to reconsider his much-quoted statement.

Since preparing this paper I have secured a copy of a report, just issued by the Medical Research Council of Great Britain, upon the work of Mrs. Mellanby, and, in the introduction, these statements occur:

As the outcome of this work it seems clear that the liability of a tooth to decay depends largely upon the protection of its structure and that this in turn is to a great extent dependent upon nutritional influences during growth, both antenatal and postnatal.

Another extract reads:

This report also supplies evidence to show that there are two main conditions which control the onset of dental caries: the better the teeth are formed the more resistant they are to caries and, independently of structure, resistance of teeth to decay is directly influenced by the constitution of the diet. The chemical agencies in food which lead to the formation of well calcified teeth are, at least in part, the same as those which increase or maintain the degree of immunity to caries shown by the teeth in later life. The knowledge available as to what foods produce well or badly calcified teeth can therefore now be applied also to make the developed teeth resist decay, and often indeed to arrest decay in teeth already attacked by the disease.

The White House Conference at Washington in 1930 stated that the nutritional effect on the teeth is regarded as being the dominant factor in dental physiology and pathology.

The following few quotations are from Mrs. Mellanby's work. She writes:

All tests made in Birmingham in 1931 showed in children that the resistance to caries could be altered significantly by slight but specific dietetic variations.

In regard to tests at Sheffield Mrs. Mellanby makes this statement:

The inference drawn from the Sheffield test is that the onset of caries in children's teeth, whatever their original structure, can be influenced by diet. Thus a diet rich in vitamin *D* and containing sufficient calcium and phosphorus inhibits the onset and spread of caries and tends to "heal up" carious areas. This inhibition is increased by the omission of cereals.

Again:

Primitive or uncivilized races, living in their natural habitat and removed from influences of civilization, are much more free from dental disease than civilized races, and when individuals or races leave their country and live under civilized conditions or remain in their own country and adopt the diet of civilized communities, the teeth become more liable to disease.

Mrs. Mellanby also proved pyorrhœa could be produced in animals by regulation of the diet.

I have avoided mention of personal findings in my own practice, but I have definitely proved that where sensible cooperation exists with the patient, simple alterations in diet play the greatest part in the cure of periodontal disease.

NOTES ON PERIODONTITIS AND ITS GENESIS.[1]

By Harvey Sutton, O.B.E., M.D., B.S. (Melbourne), D.P.H. (Melbourne), B.Sc. (Oxon).

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The aspect of dental disease to which this paper is devoted, concerns the supporting tissues of the teeth—the gum, the periodontal membrane and the cementum. It is the most ancient form of dental disease and is still prevalent in civilized adults.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and of the New South Wales Branch of the Australian Dental Association on August 30, 1934.

Elliot Smith, in his study of Egyptian mummies,[1] has shown that in the jaws of mummies dating from the predynastic period before the pyramids were built (? 3500 B.C.), the teeth show marked attrition, thanks to the grit of the grinding stones present in the meal. Secondary dentine is formed, but caries is absent. Periapical infection and alveolar infection are quite common.

Among the nobles, and increasingly as civilization advanced among the people at large, caries became common, for civilization brought with it the consumption of excessive amounts of purified flour, as is the case today. The distinction between caries and alveolar disease is here well defined, and indeed pyorrhœa may be present, yet the teeth prove actually resistant to caries.

Periodontal disease is a general term (like paradentosis) applied to a series of changes occurring around the roots of the teeth below the enamel level.

The tissues of the gum, the periodontal membrane and the cementum surrounding the root are the central point of attack. In advanced changes absorption of the alveolar bone of the tooth socket occurs with loosening of roots and loss of the teeth.

The earliest stage is gingivitis. Soon the "rolled-over" gum, swollen, easily bleeding with contact with the tooth brush, becomes the front wall of a pocket or sinus which develops in between the gum and the cementum.

In this pocket *débris* of food collects, bacterial action becomes marked, with deposit of tartar and even with the production of whitish or yellowish discharge; hence the term *pyorrhœa alveolaris*.

The "rolled-over" gum is not rare in school children, but even in early adolescence pockets and pyorrhœa are distinctly uncommon. Yet in young men in the early twenties the condition of early pocketing is quite frequent in the lower incisors. Osler used to draw a visiting card in between these teeth and point out the characteristic cheesy odour, ranging from the mildest cream cheese to the most advanced Gorgonzola. The odour can be reproduced in cultures made of the organisms in the pockets.

Examination of the pockets even at this early stage shows that epithelialization has taken place, the stratified epithelium of the outer surface of the gum having grown down to line the external wall of the pocket, thus being responsible for its persistence. The swollen gum margin and the enlargement of the interdental papilla are typical.

The prolonged insidious progress of the condition is characteristic. Although the disease may be evident in the twenties, the loosening of the teeth may not occur till twenty or more years later.

Attention may be drawn to the time factor in the development and eruption of the tooth. This is all important for an understanding of the possible influence of vitamins and of the whole course of the disease.

We may pass over the story of the deciduous teeth, which, as already explained, do not come into the picture.

The dissection of the jaws of children and the time-table of the eruption[3] furnished by the Medical Research Council gives us the facts as to the period of development of the cement and roots of the permanent tooth (see Table I). Whereas the

TABLE I.

Eruption of Teeth.

(Adapted from Medical Research Council Report, Special Series, Number 97.)

Tooth.	50% Present. (Age in years.)		50% Fully Erupted. (Age in years.)	
	Boys.	Girls.	Boys.	Girls.
1 | 1	7·4±0·5	7·2±0·5	8·7±0·6	8·4±0·6
1 | 1	6·5±0·5	6·2±0·5	7·7±0·6	7·9±0·6
2 | 2	8·8±0·7	8·4±0·7	10·1±0·8	9·9±0·9
2 | 2	7·7±0·5	7·5±0·5	8·8±0·7	8·7±0·6
3 | 3	11·7±0·8	11·2±0·8	12·8±0·8	12·3±0·8
3 | 3	10·8±0·8	9·9±0·7	11·9±0·8	11·0±0·8
4 | 4	10·0±1·0	9·8±0·8	10·8±1·0	10·7±0·9
4 | 4	10·9±1·0	10·4±0·9	11·7±0·9	11·3±0·9
5 | 5	10·9±1·1	10·7±1·0	11·4±1·0	11·2±0·9
5 | 5	11·8±1·1	11·2±1·1	12·4±0·9	11·9±0·9
6 | 6	6·3±0·5	6·1±0·5	6·9±0·5	6·7±0·6
6 | 6	6·2±0·5	5·9±0·5	6·8±0·5	6·6±0·6
7 | 7	12·3±0·8	12·1±0·8	13·0±0·8	13·0±0·8
7 | 7	11·9±0·8	11·5±0·8	12·6±0·7	12·3±0·8

enamel of the crown is built in the first few years of life, very little of the root is in existence until eruption has actually commenced. Hence if we presume that dietary decides the enamel structure, this is the function of the years up to five. Whereas, if dietary is to influence the structure of the gums and cement, later years are more important. The eruption takes, from the appearance through the gum till complete eruption and alignment and apposition to its pair of opposing teeth, a period of about one year or more.

Before the age of seven years scarcely any of these tissues are fully formed (see Figures I, II and III).

Figures I, II and III are drawings from the sections of the jaws of children by kind permission of Professor Burkitt, Anatomical Department, University of Sydney. Three age periods are shown: Figure I, just after the sixth birthday; Figure II, about the seventh birthday; Figure III, after the tenth birthday. Only in Figure III are the beginnings of the roots of unerupted permanent teeth in existence.

Obviously it is the dietary during school life, six to eighteen years, to which we must look for proper supplies for periodontal tissue formation.

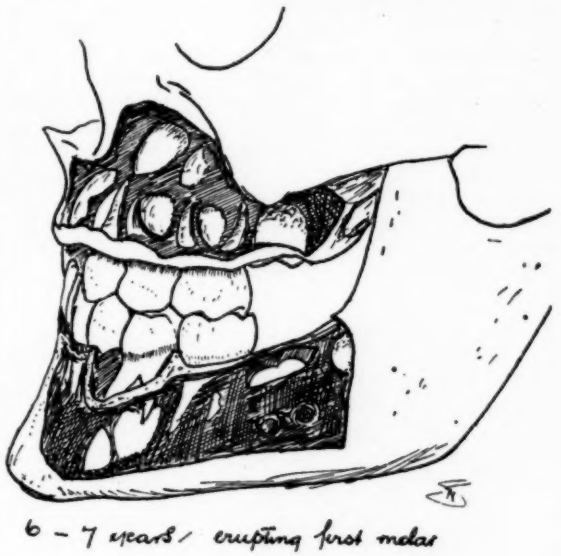

FIGURE I.

As the enamel organ pushes through the gum, the Nasmyth membrane carries with it the superficial epithelium, so that about one-third of the lower part of the crown is still covered, and only after many years does it retract in the normal tooth to the

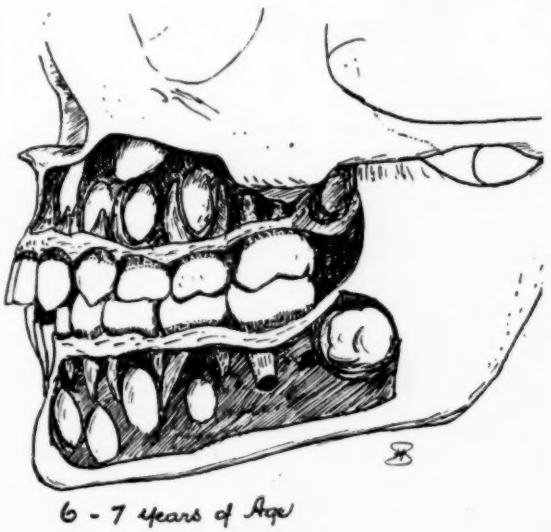

FIGURE II.

cemento-enamel junction.[9] Here already a potential pocket is present, and the gingivitis produced by the Vincent organisms is very common in young adult life.

In over 4,000 children in England, up to 60% at the age of thirteen show definite, though slight, gingivitis, while chronic marginal gingivitis is common in mouth-breathers.[4]

I have seen definite epidemics of rolled-over, swollen, engorged gums in institutions. One such was due to the use in common of tooth brushes. Kirkpatrick, in one hundred patients, chiefly adults, records practically 100% at twenty years of age with spirochætes. Inflamed gums rapidly rise in frequency at twenty to a maximum at thirty-five, and periodontitis with pockets at twenty-five, with a maximum at forty-five years of age. The gingivitis due to Vincent's infection is thus strongly associated with later pocket formation.

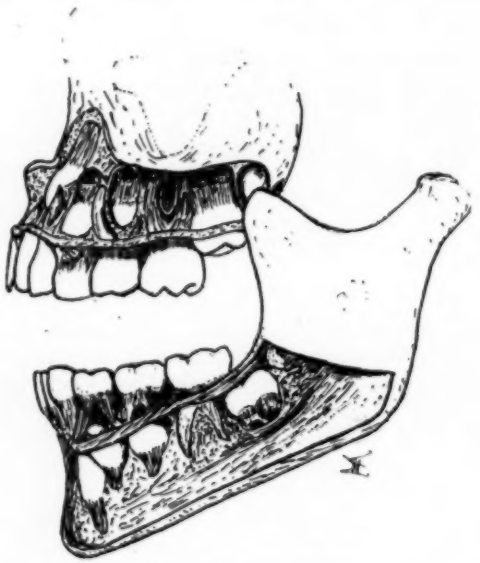

Figure III.

Mrs. Mellanby[2] (1930) carried out an extensive and carefully controlled series of animal experiments, chiefly on dogs.

The structure of the hard dental tissues and of the alveolar bone were demonstrated to be susceptible to dietetic influences.

Provided that a certain minimum of mineral salts, calcium and phosphorus, were present in the diet, perfect structure could be insured by adequate supply in the diet of vitamin *D* (calciferol) or under certain conditions of irradiation of the animal.

Insufficiency of vitamin *D* meant imperfect tooth structure. Cereals, especially oatmeal, and to a less degree white flour and rice, tended to hinder calcification, but the harmful effect was readily neutralized by sufficiency of vitamin *D*. Rabbits, rats and man himself show similar results.

Secondary dentine in response to attrition is, of course, due to the power of the osteoblasts of the pulp lining the inner dentinal surface to display renewed activity. This showed a corresponding relationship to sufficiency or deficiency of vitamin *D*.

On the other hand, vitamin *A* appears to be responsible for the sound structure of the soft tissues, just as vitamin *D* controls the structure of the hard tissues. Deficiency of vitamin *A* shows itself in keratinization of skin or mucous membranes, together with hyperplasia and metaplasia. "Dry eye", septic sinuses, indeed sepsis anywhere, demonstrate a failure in resistance against microbic invasion. Sections of human gum excised during treatment of pyorrhœa[10] show either definite keratinization of the epithelium with but little round-celled infiltration, or else engorged blood vessels with definite infiltration by round cells without epithelial changes. Both probably represent a weakened attempt to establish a defence against infection. These epithelial changes are of particular interest with regard to recent work in Sydney concerning tropical ulcer and New Guinea sore mouth.

Injury of the gums in dogs may start up local disease.[2] Diet during development will still decide the presence of resistant structures and the later diet also affects the progress of the disease; for example, oatmeal, as part of a deficient diet, favours tartar deposit and later periodontal disease; nevertheless abundance of carbohydrate in a properly arranged diet does not produce disease.

Periodontal disease could be produced or prevented in dogs. If a puppy's diet has been deficient in vitamins *A* and *D* during the early months of life, that is, when teeth or periodontal tissues are developing, hyperplasia of the subgingival epithelium develops with vitamin *A* deficiency and invasion by microorganisms follows. Where vitamin *D* is similarly deficient the adveolar bone shows defects.

If this dietetic failure occurs during the development of the teeth and periodontal tissues, the dog is liable to develop periodontal disease at a later stage, whatever the subsequent diet.

When the diet is properly arranged in the developmental period, the occurrence of periodontal disease at a later stage is prevented or greatly inhibited even though the adult diet is deficient in these vitamins *A* and *D*.

The exact description of the gingival margin appears to vary with different observers. Probably, in the ideal normal arrangement, the gingival epithelium ends flush with the tooth at the enamel-cement junction, really being continuous with Nasmyth's membrane covering the enamel. In the majority,[11] however, the gum extends for a short distance over the enamel, forming a minute sulcus with a subgingival layer of epithelium growing down to the level of the enamel-cement junction. Here it is interrupted by the strong bands of fibrous tissue which extend out from the cementum, reinforced by the fibres of Sharpey, which act for the layers of cementum and of bone much as the sprigs do for the leather layers in the sole of a boot. These bands unite teeth together and, passing in varying direc-

tions down and up the periodontal membrane, they sling the root of the tooth in suspension, much as earthquake houses in Japan are slung on chains to meet these vibratory shocks.

Obviously, from a mechanical point of view, the essential patents in the dental machine are properly developed enamel, free from defects, and powerfully built supporting suspension or shock absorber apparatus (cement, periodontal membrane and gums). The vulnerable points are the epithelium of the gums of the gingival margin, the attachment to the enamel-cement junction, and the thin absorptive layer of cement lying between the enamel and the alveolar margin.

The whole dental apparatus is profoundly altered in its growth and development by lack of the fat-soluble vitamins *A* and *D*.[2] The gingival margin thickens, the knife edge becoming a rolled-over gum tending to leave a trough between gum and tooth, while its attachment to the badly built, rough and pigmented (hyperplastic) enamel is imperfect. The subgingival epithelium increases in amount with finger-like processes extending down over the cement. This growth of epithelium is probably an epithelialization of the deep surface of the gum and lines the sac or pocket which extends a variable distance along the tooth, leaving an increasing amount of cementum bare (see Figure IV).

The main tissues of the gum (corium) show considerable cell infiltration amongst the tough fibrous tissue, representing apparently a partial failure of its defensive immunity against infection.

The periodontal membrane is irregular in thickness, the alveolar bone is less dense, osteoid tissue and poorly calcified bone being seen in the alveolar crest; and the *lamina dura*, the bone trabecula, are lacking in definition and therefore lacking in their resistance to stress and strain, which these bony struts are constructed to meet. Hence wear and tear will more rapidly weaken their support and irregularities in alignment and occlusion tend to be more common.

Of special interest have been the recent investigations of Dr. F. W. Clements, of the School of Public Health and Tropical Medicine, and R. M. Kirkpatrick, Dental Research Scholar, who are continuing their work at Manus, one of the islands of the mandated territory of New Guinea. The presence of fourteen Papuan native students at the school gave the opportunity of studying the condition of New Guinea mouth, which they all presented.[7] This was diagnosed as Vincent's infection (ulcero-membranous gingivitis).

Orange juice is found to be of value. All the fourteen received the same diet. Four were kept as controls. Additions to the diet were made as follows. Four received orange juice (vitamins B_2 and *C*), four received treated orange juice (vitamin *C*), two received yeast extract (vitamin B_2). Yeast extract caused marked improvement, as also did the orange juice in larger amounts. Vincent's infection evidently reacts favourably to vitamin B_2.

Tropical ulcer is another form of infection by the Vincent organisms. It is preceded by keratinization and hyperplasia of epithelium. Epidemiological evidence suggests that diet has a controlling effect on its district incidence. Presuming that a vitamin deficiency has a predisposing, and a full supply of vitamin a preventive influence in regard to the disease, the hypothesis remains to be further tested and the vitamin identified.

It is interesting to recall the curious discrepancies in the signs of scurvy among young children and in adults. In babies fed on boiled milk scurvy readily develops with typical bony and periosteal changes, but the gum condition is absent, whereas in adult scurvy the spongy, bleeding gums are frequently observed. The question arises, as Clements points out, whether the gum condition is a true manifestation of vitamin *C* deficiency or scurvy, or is due to another vitamin. Such combined deficiencies are, of course, not at all rare, for example, vitamins *A* and *D*, *C* and *D* (scurvy, rickets *et cetera*). It is also interesting to note that the age for spongy gums corresponds to the

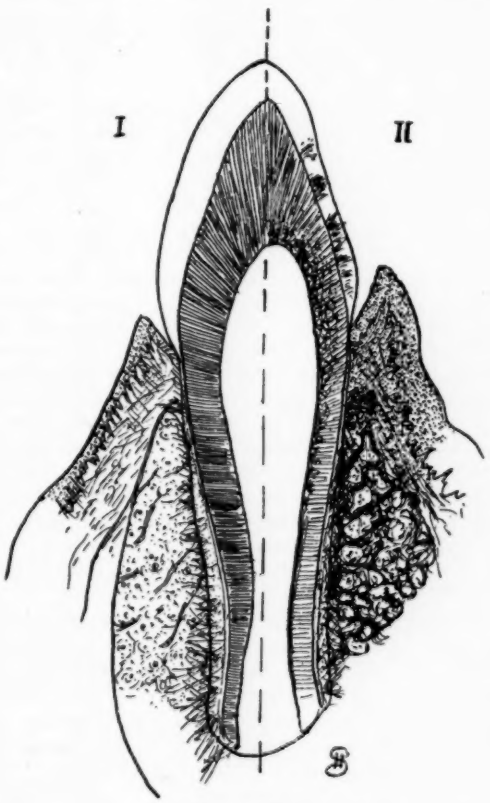

Figure IV.

Diagrammatic section of normal (I) and deficient (II) teeth (after Mellanby). Thick gums, thick and irregular epithelium, round-cell infiltration, poorly calcified alveolar bone with osteoid tissue, wide irregular periodontal membrane, dentine thinner with interglobular spaces, enamel stained. Deficiency in vitamins *A* and *D*.

age for pyorrhœa, and in the very earliest description (1746)[8] the association was noted as well as the failure of antiscorbutic treatment for the latter.

These two young researchers have thus opened up a new field, in which New Guinea mouth—tropical ulcer[6]—and *pyorrhœa alveolaris* are all linked together, the effects of a definite infection in tissues predisposed by a certain (at present undefined) vitamin lack. It is fairly certain that neither vitamin *C* nor vitamin *D* is concerned. Vitamin B_1 does not come into the picture. The decision has yet to be reached as to whether it is vitamin B_2 or vitamin *A*, probably the former. Should this be definitely confirmed, the value for the treatment of pyorrhœa should be of real significance.

A brief history of the hypotheses regarding these conditions is manifest in the various names given to the condition.[8]

In 1746 Fauchard described the gum condition as a variety of scurvy which he found resistant to ordinary antiscorbutic treatment, and in 1778 Jourdain separated this syndrome from scurvy, claiming, however, an association with rickets; while in 1786 Bourdet pointed out that it was not specially associated with rickets, Toirac (1823) described it as interalveolodental pyorrhœa, Oudet (1835) as pericementitis and chronic purulent periodontitis. In 1845 Riggs, of Connecticut, drew public attention to the condition, and it was often called Riggs's disease. Thomas, in 1850, called it chronic gingivitis, while de Calvi considered it to be expulsive gingivitis. Since then (1873) we get alveolodental periostitis (Magitôt) and alveolodental arthritis (Joyeux). Rehwinkel, in 1877, following European ideas, read a paper in the United States of America on *pyorrhœa alveolaris*, which is the commonest name in English. It is not really satisfactory, for pus production is not necessarily an essential association.

In the last ten years we meet with another series of names — spirochætosis, arthrodental infectious gingivitis, alveoclasia, dental polyarthropathy, peridental disease, periclasia, alveolysis, alveolodental pyorrhœa, atrophic *senilis præcox*, alveolar pyorrhœa, not to mention *Schmutz pyorrhoe* and *Zahnbettschwund* (atrophy of the tooth socket). While the favourite names today appear to be periodontoclasia (America) and paradentosis (Continental), a journal, *Paradentosis*, is now devoted entirely to this disease.

We find, too, increased classification of causes into centripetal and centrifugal, exogenous and endogenous, and every possible process has been brought into causal association according to the fashion, such as vitamins, endocrines (for example, in diabetes), allergy.

In this paper stress has been laid on the phenomena of growth and development of the teeth, the vitamins necessary for resistant structures, the importance of gingivitis in the genesis of the condition, and in general the time factor in its causation and progress. Proper consideration of these facts will place many alleged causal factors in their true relationship. For example, great stress is laid on occlusion by many dentists. It is interesting to note that the whole structure of the tooth, the crown, the root, the alveoli and the gums are laid down in complete structure before occlusion occurs.

Whereas the condition is very common in the lower incisors, it is far less common in the upper incisors which meet them in occlusion. It is difficult to see why, if occlusion dominates the problem, in two groups of teeth the occlusion of which may be mutually unsatisfactory, one group should display disease and the other group escape.

It is interesting, too, to find in the jaws of prehistoric primitive man, whose teeth are in excellent alignment and literally ground down to the roots, the presence of alveolar disease.

Confusion has arisen probably because *débris* and tartar readily collect around any unused tooth, for example, a tender tooth, and prepare the conditions in the gingival pocket which lead to infection.

Dentures and other artificial appliances may be associated with pyorrhœa again by the accumulation of *débris*, but the condition is found in thousands of people without dentures at all.

No doubt various occupations, that of salt workers *et cetera*, may show gum ulceration and loss of teeth, and various constitutional conditions, diabetes and gout, be commonly associated. This is, however, of minor importance, for the condition is chiefly present in large numbers of young adults unaffected by these factors.

Endocrine effects have not been touched on, and no doubt vitamin action is intimately concerned with endocrine action. The evidence points to the action of these glands as secondary and not causative in this condition.

To concentrate, as so many do, on what is found in older adults is to deal with complications and sequela and to miss the essential factors responsible for the origin of this disease many years previously.

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THE ÆTIOLOGY OF DENTAL CARIES.[1]

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A tooth consists of a crown and a root. The crown is external to the gingivum and is made up of an outer covering of enamel, a middle mass of dentine and a central small portion of pulp. The root is internal and consists of an outer covering of cementum, a middle mass of dentine, and a mass of pulp into which, at the apex of the root of the tooth, pass the blood vessels, lymphatics and nerves. All parts of the tooth are important in a consideration of diseases of the tooth, but as this paper has to do with the ætiology of caries, we shall consider only the enamel in detail, for it is now almost generally agreed that caries is primarily a disease of the enamel.

Enamel.

The enamel is epiblastic in origin. It is unique in being the only calcified structure that is not mesoblastic in origin. In keeping with its origin, it is in its development more likely to be affected by those diseases that affect the epithelial tissues (Noyes[50]), for example, the exanthemata and especially scarlet fever; and a priori one would expect that lack of vitamin *A*, which, as McCollum[31] stresses, affects chiefly epithelial tissues, would have a more marked effect than lack of the other vitamins. However, we shall consider the actual effect of vitamin lack later. The enamel of the tooth is complete before its eruption and though, as Pickerill[51] has shown, it may increase in density subsequent to eruption, this hardening process, according to modern views in crystallography, would be due to intramolecular rearrangement and to a progressive keratinization of the processes of the ameloblasts which are left surrounding the enamel prisms. In other words, the increase in density does not result from any so-called vital or nutritional process. Histologically, enamel is seen to be composed of crystalline prisms or rods arranged in a more or less parallel manner. Around each rod is a small amount of protein of a keratin nature. There is evidence that enamel is to some extent porous (Beust,[3] May Mellanby,[49] Pickerill[51]). Allen[1] goes so far as to describe a most elaborate system of tubules arising in the dentine and passing into the enamel, but it is generally agreed by almost all dental histologists that there are no nutritional channels pervading the enamel; and all modern attempts to inject human enamel from the pulp cavity have failed (Noyes,[50] Pickerill,[51] Hopewell-Smith,[20] Leon J. Williams,[45] Gies,[14] Beust[3] and Chase[10]). Fish,[48] who for long maintained that enamel was completely porous and nourished from the dentine, has recently reversed his opinion and now is in complete agreement with those who consider the enamel impervious. It is fundamental to the problem of dental caries to be quite clear on the question of the permeability and nutrition of the enamel. After surveying the literature, we feel that there is unassailable evidence to conclude that erupted enamel is an inert crystalline mass which has no blood supply and no circulation, and which is quite independent of any nutrition. Erupted enamel therefore cannot be influenced from within by foodstuff, vitamin, salt or any essential substance. No one has demonstrated any repair of the enamel. Allen[1] says that he frequently notices enamel repair, but fails to illustrate it. We were unable to find any other reference to such a phenomenon. It is true that certain portions of the enamel may be caries-resistant, but this has almost definitely been proved to be due to the deposition of a complex organic plaque of mucin, dead bacteria *et cetera* which is acid-resistant and which protects the underlying enamel.[12] When caries becomes arrested, the enamel has already been destroyed and the arrest is due to changes in the dentine, which is a living tissue and is able to produce a calcified material. The ætiology of caries is essentially comprised in those factors that lead to a destruction of enamel, and is not concerned with the reparative properties of the dentine. Though it is well known that even perfect enamel is susceptible to decay, it is natural to consider that, other things being equal, and quite independent of the cause of caries, a properly developed enamel of maximal density and uniform composition will offer greater resistance to disease than a thin enamel of a hypoplastic nature, so that it is important to study the factors influencing the composition of developing enamel.

Factors Influencing the Composition of Developing Enamel.

The following factors have been stressed by different groups of investigators as having an influence on the developing enamel: calcium and phosphorus metabolism, the vitamins, illnesses.

Calcium and Phosphorus Metabolism.

In many institutions for children the calcium and phosphorus of the diet have been shown to be decidedly below the so-called optimal minimal requirements, yet the teeth of such children are in general almost caries-free (Bunting,[8] Waugh,[44] McBeath[30]). The calcium and phosphorus content of the serum of children is remarkably constant over a wide range of calcium and phosphorus intake and type of dietary (Bunting,[8] own studies[16]), and as the enamel of the tooth develops slowly and requires comparatively little of the available calcium and phosphorus, it should have little difficulty in competing successfully for its requirements, and so the availability of calcium and phosphorus is not as important for the developing enamel as for the rapidly growing bones. Dental caries affects up to 95% of the population and the incidence has

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and of the New South Wales Branch of the Australian Dental Association on August 30, 1934.

increased during this century, but the total intake of calcium and phosphorus in the food during the last few decades has certainly not decreased. There is a great deal of clinical evidence that calcium and phosphorus deficiency is not of any moment in determining hypoplasia of the enamel or an increased susceptibility to caries. Mackay[27] studied the teeth of children who had had clinical rickets and found that the enamel, which was being formed at a time when rickets was present, was not abnormal. Bloch[5] reported similar findings. The diet of the Eskimos, who have excellent teeth, is poor in calcium and rich in phosphorus (Waugh[43]). Phosphorus deficiency has been claimed to determine enamel hypoplasia (Klein[25]). The evidence is not at all convincing, as pointed out by the Pittsburgh research workers.[12] Sherman ("Chemistry of Food and Nutrition"[52]) states that not more than 1% of American diets of caloric value sufficient to meet growth requirements would be deficient in phosphorus. On the other hand, Hoppert and co-workers,[21] and McCollum and Klein[25] point out that caries may occur in the rat even though the diet during development of the enamel has been adequate in calcium and phosphorus. There is ample evidence to conclude that children on an average mixed diet of adequate caloric value cannot have a deficiency of calcium or phosphorus of a degree that would determine hypoplasia of the enamel of any significance, so that in general calcium and phosphorus deficiency is not of any ætiological importance in dental caries.

The Vitamins.

It was inevitable that the vogue of vitamins should spread to the problem of dental caries. Various research workers found that experimental animals, deprived during growth of one or other vitamin, showed defects of various parts of the tooth or periodontal tissues. There is no doubt that in the complete absence of vitamin *A*, *C* or *D* the teeth of various laboratory animals, and especially the guinea-pig, show severe lesions,[17] but on the available evidence there does not seem to be in the human being a corresponding relationship between vitamin lack and dental defect.

Vitamin A.—Bloch,[5] of Copenhagen, reported the condition of the teeth of a series of children whose deprivation of vitamin *A* during the period that the permanent teeth were developing had been so severe that xerophthalmia leading to permanent blindness resulted. He states:

> In man, deficiency of vitamin *A* has no specific injurious effect on the formation and calcification of the teeth. A disposition to dental caries therefore cannot be due to deficiency in vitamin *A* in infancy.

If extreme vitamin *A* deficiency had no effect, one can reasonably conclude that hypothetical deficiencies in the range of diets eaten by infants of civilized nations cannot account for a 95% incidence of dental caries.

Vitamin C.—Deficiency of vitamin *C* as a cause of dental caries has many advocates (Höjer,[19] Eddy,[11] and Howe[22]). Hanke[17] insists that a pint of orange juice should be taken every day to provide sufficient vitamin *C*, but again evidence against the importance of vitamin *C* being a causal factor is overwhelming. In Hawaii,[9] where there is a large consumption of fruits rich in vitamin *C*, caries is rampant. McCollum[31] points out that the diet of Eskimos is very poor in vitamin *C*, yet caries is very rare. He also states that Cilento informed him that the natives about the island of New Guinea consume bananas, which are rich in vitamin *C*, yet caries is common. On the experimental side, McCollum states that marked caries can be induced in rats, an animal that is able to synthetize vitamin *C*. He writes:

> I cannot see how the extreme views of those who place all the emphasis on the antiscorbutic vitamin can be justified.

Vitamin D.—We now tread on dangerous ground, but we are not alone in doubting that deficiency of vitamin *D* during the development of the teeth is the cause of the prevalence of dental caries. Everybody appreciates the enormous amount of work May Mellanby has done in an attempt to prove, first, that vitamin *D* deficiency during the development of the teeth leads to hypoplasia of the enamel, which, she claims, is a potent contributing cause of caries, and, secondly, that the administration of vitamin *D* after eruption of the teeth will determine a healing of caries. It is true that in experimental animals a gross deficiency of the calcifying vitamin leads to defects in the developing teeth (Blackberg,[4] Shelling[37]), but such extreme experimental conditions do not obtain in human beings. May Mellanby's conclusions have received a great deal of criticism. Sim Wallace[40][41] has marshalled a great deal of evidence against the importance of vitamin *D*, while the clinical studies of the teeth of rhachitic children by Mackay,[27] Bloch,[5] Hess[18] and Sprawson[39] show no relation between dental caries and the rhachitic diseases of bone, which can, of course, be caused by a lack of vitamin *D*. Others to criticize her work are C. Mann,[29] Gies,[14] Rosebury,[36] Enright, Friesell and Trescher,[12] and Osborne.[34] Briefly, these authors show that there is overwhelming evidence that dental caries may exist though there has been no lack of vitamin *D* during the development of the teeth, that decay may continue though large doses of vitamin *D* are being administered, and that hypoplastic teeth do not necessarily decay.

Illnesses.

There is some evidence that the exanthemata occurring during the development of the teeth may determine changes of a hypoplastic nature in the form of bands and milk spots [Mellanby,[49] Haggett (personal communication)].

Fluorine.—There is a condition known as "mottled enamel" which is endemic in certain parts of the United States of America,[33] of China and Sicily. This mottling has been proved to be due to the presence of fluorine in a concentration of two milligrammes or more per litre in the drinking water. Most waters have from nil to 0·3 milligramme per

litre. Fluorine is a normal constituent of enamel. Mottled enamel is disfiguring, but apparently does not render the teeth more liable to caries (Bodecker[6]), but Koehne and Bunting have very recently produced evidence that it does.[46]

Work at the Royal Prince Alfred Hospital.

During an investigation of one hundred diets of normal children between the ages of four and fourteen years an attempt was made to correlate the condition of the gums and teeth with the following factors: local care of the mouth, early history of the child as regards health of mother during pregnancy, exhibition of vitamin concentrates in early childhood, amount of calcium and phosphorus in the diet, amount of easily fermentable carbohydrate in the diet. We have not yet gone fully into our results, which will be published later. Such studies are not a satisfactory means of investigation. A detailed anamnesis and examination do not give an adequate life history of the child; they do not discover to us the ultimate effect on the teeth of those factors that are still functioning or those that have ceased to function, for we must remember that the incidence of caries falls many years after all the factors that may influence developing enamel have ceased to operate. Secondly, it is impossible to draw scientific conclusions when we have a number of factors none of which is controlled. Thirdly, the dentition between the ages of four and fourteen is extremely unstable, and the amount of caries may change rapidly with changes in the number of deciduous and permanent teeth. Fourthly, some degree of caries was universal. In such a study negative correlations may be permissible, and there seems sufficient evidence to conclude that adequate supply of vitamins, calcium and phosphorus, good physical development of the child or good health of the mother during pregnancy does not protect the child against caries. The history of one family is instructive.

The five youngest members of a family of ten were examined. The parents are and have been for years in poor circumstances. The diet was low in calcium and phosphorus, the children had practically no fruit, had never had any vitamin supplements, but they had very little cake and practically no sweets. The dentition of each child ranked amongst the very best of the whole series.

Summary of Factors Influencing Development of Teeth.

Dental caries affects all grades of the social scale and all civilized societies. In an average mixed diet of adequate caloric value for growth requirements there will be sufficient inorganic salts to provide the developing teeth with their needs, and the common foodstuffs *plus* the sunshine in temperate zones provide sufficient vitamins *A*, *C* and *D* to enable these salts to be adequately utilized. The available evidence indicates that neither errors of calcium and phosphorus metabolism nor lack of adequate vitamin are playing any essential part in the causation of caries. In thus summarizing this section of the paper it must be clearly understood that we are concerned only with dental caries and not with the effects of deficient diet or vitamin lack in other disorders of the teeth or gums or other parts of the body, or with the advisability of giving vitamin supplements to children.

The Mechanical Theory of Caries.

Maddern[28] has recently developed a "mechanical theory of the causation of caries". A careful perusal of his latest publication is not convincing. A great deal more fundamental work and more mathematical data are required before such a theory could receive serious consideration.

The Chemistry of Enamel and Saliva in Relation to Dental Caries.

The primary lesion of caries is the passage into solution of the surface of portion of the enamel. So long as the enamel is intact there is no decay. Enamel has the following composition (Noyes[50]): inorganic matter, 98% to 99%; organic matter, 1% to 2%, the principal elements being calcium, phosphorus, fluorine and magnesium.

The predominant salt is tricalcium phosphate, $Ca_3(PO_4)_2$, which is a very insoluble salt. In the mouth we have the enamel surrounded by saliva, which has frequently been proved to contain tricalcium phosphate in a concentration much greater than that which could be held in simple solution, and this concentration of tricalcium phosphate is not different in the saliva of carious or non-carious mouths (Forbes,[13] Benedict and Kanthak,[2] Enright *et alii*[12]). Changes in the concentration of hydrogen ions, that is, changes in acidity or alkalinity of the saliva, determine whether more calcium phosphate can be held in solution or whether precipitation will take place. If local conditions are such that the alkalinity of the saliva is increased, we may get the deposition of calcium phosphate as tartar. This phenomenon has been correlated with the presence of a phosphatase by Smith.[38] If the acidity is increased, we get solution of the enamel (Benedict and Kanthak[2]). Experimentally, the work of Enright, Friesell and Trescher has shown that enamel is dissolved at a pH of 5, even in a solution buffered with tricalcium phosphate, but, of course, the general bulk of the saliva is never as acid as this; indeed, as the Bunting research group has shown by analysis, specimens of saliva from children both immune from and susceptible to caries show no significant differences in reaction. If the saliva reached an acidity of pH 5 the whole surface of the enamel would be disintegrated, and especially that portion of the surface which is continually bathed by saliva. Acidity of the saliva as a whole, therefore, can play no part in the problem. But when we focus our attention on the special conditions obtaining at sites where caries begins, that is, in areas protected by anatomical peculiarities, by bacterial plaques or by the retention of food material, it can readily be conceived that in such places the hydrogen ion concentration might increase to a pH of 5; and the onset of caries could be readily explicable in terms of simple chemical considerations if it could be proved that there

existed in the mouth a mechanism by which a pH of 5 could be locally attained. This brings us to the bacterial theory of dental caries.

The Bacterial Theory of Dental Caries.

The bacterial theory of caries is by no means recent, and organisms were found in decaying dentine as early as 1881 by Miles and Underwood, of London, while W. D. Miller, working with Koch, isolated from carious dentine, organisms that produced acid fermentation. However, it was left to the careful bacteriological studies by Rodriquez,[35] McIntosh[32] and Jay[23] to bring out the relative importance in the production of caries of the various types of bacteria found in the oral cavity. As a culmination of these bacterial studies, Enright, Friesell and Trescher, and Bunting and Hadley have established almost unassailable proof that a specific organism, *Bacillus acidophilus*, is capable of producing and existing in an acidity of pH 5. This organism has been shown by careful quantitative work to be present in large numbers in the saliva and in scrapings from carious areas in the mouth of those affected by caries, and to be present in relatively small numbers in or absent altogether from caries-immune individuals. If it is present in large numbers at a time when caries is absent, a follow-up of such patients shows that caries has now commenced. Other organisms were shown to be unimportant in as much as they have been unable to produce acid or even live in an acid medium. Bunting and co-workers[8] showed that in certain caries-immune individuals it was impossible by feeding cultures of *Bacillus acidophilus* in enormous numbers to establish this organism either in the mouth or bowel. In other words, there is in some individuals an immunity mechanism, which is apparently due to the presence of specific antibodies in the saliva and bowel, but so far the factors that enhance or decrease the potency of this mechanism have not been elucidated. Further research is urgently needed.

In this brief account it is impossible to do justice to the excellent research that brought order out of chaos and firmly established the true bacteriology of dental caries. To gain an adequate appreciation of the work it is necessary to consult the original papers.

Pabulum of Bacillus Acidophilus.

The next aspect of the problem is the relationship of *Bacillus acidophilus* to the type of food eaten. It has been abundantly established that the media most easily utilized by *Bacillus acidophilus* for the production of acid are the monosaccharides and the soft, well cooked starches. When sugar or starch is given in such a form that it provides a sticky mass, such as chocolate, sticky sweets, sweet cakes or sweet biscuits, it will adhere to protected sites and provide ample pabulum extending over many hours for the rapid conversion into lactic acid. Beginning with the work of Miller in the last decade of the nineteenth century, a great many workers have stressed the part played by a suitable pabulum, amongst the most noted early contributors being Black, Leon Williams and Pickerill. In the last few years great emphasis has been placed on the pabulum of *Bacillus acidophilus*. Recently, Waugh[42] said:

> I should like to allude briefly to the study I have made of the changing diet of the American Eskimo and the resultant effect upon his teeth. Both in Labrador and in Alaska I found that the old Eskimos who in childhood had lived on the primitive native foods of their ancestors, even though of small stature, had very large, well formed and regular jaws, and decay of the teeth was almost absent. It was a common experience to find all of the teeth, without one carious cavity, present in very many mouths. They were invariably much worn on the biting surfaces, often having been reduced almost to the gum line . . . . In the twelve principal native settlements studied . . . the proteins varied from 25 to 60 per cent.—five of them being over 40 per cent. The fats varied from 25 to 66 per cent.—eight being over 40 per cent. The carbohydrates varied from 15 to 34 per cent.—four being over 25 per cent. The impressive and outstanding fact was that, in proportion as the children were fed on the sugars and starches, their teeth were badly decayed. Conversely, in the settlements where they had little or none of these foods they had very little or no dental caries.

On the experimental side, Bunting and his school were able, by adding sugar to the diet of relatively caries-immune children, to induce active caries, and by cutting down the carbohydrate of markedly susceptible individuals to reduce the number of *Bacilli acidophili* from 1,800,000 organisms per cubic centimetre to 100 per cubic centimetre. Rodriquez has achieved the same result by the same means.

It is frequently stated that pyorrhœa and oral sepsis may be present to a marked degree and yet caries may be absent, but this is not an argument against the bacterial cause of dental caries, though many have been unduly influenced by such findings. It simply means that the organisms responsible for pyorrhœa and periodontal lesions are non-acid-producers (Jay[23]). The *Bacillus acidophilus* is apparently as specific in its cause of dental caries as any other organism causing a disease entity. Apart from its ability to produce and live in an acid medium, it is a harmless saprophyte. Indeed, its presence in the bowel is often encouraged for therapeutic reasons by the administration of various sour milk preparations.

Collateral Support for Bacterial Theory.

There is certain collateral support for the bacterial theory.

Sites of Election of Caries.—There would appear to be more general agreement as to the sites most liable to caries than in any other aspect of the problem. In short, decay may begin in any area protected from the scourging action of mastication or of the lips and tongue, and in deep fissures and pits. Bossert[7] studied the upper right permanent molar in one hundred humans between the ages of seven and twenty-five years and found that the liability to caries was directly proportional to the steepness of the sides of the valley made by the cusps, that is, directly with the facility for the retention of food particles. Extracted teeth attached to plate bases and pulpless teeth show no greater

ILLUSTRATIONS TO THE ARTICLE BY DR. J. LEON JONA.

FIGURE I.

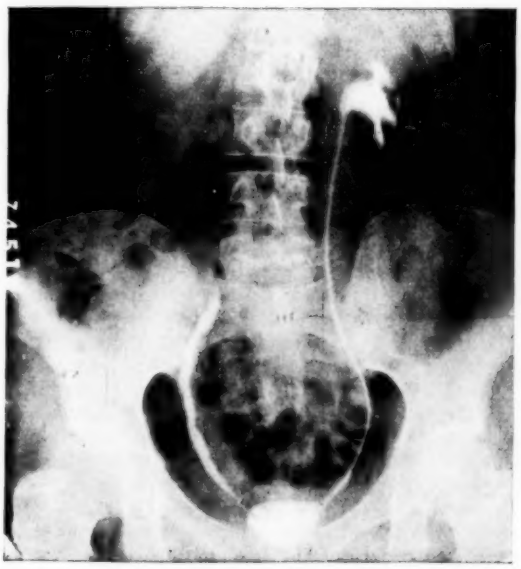

FIGURE II.

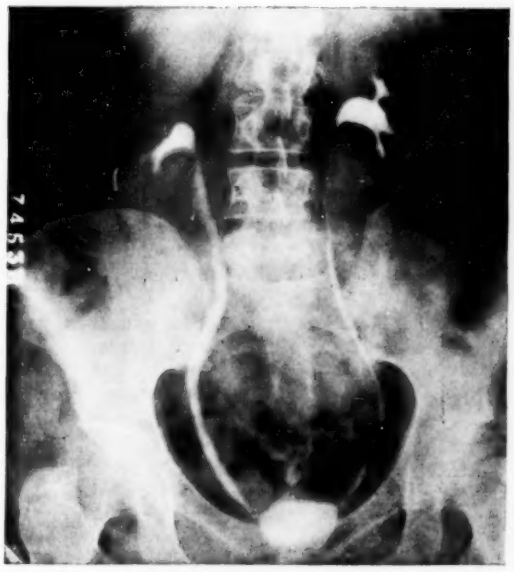

FIGURE III.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. LEON JONA.

FIGURE IV.

FIGURE V.

tendency to caries than live teeth, and caries in such teeth follows the same general lines as in live teeth occupying the same relative position.[12]

Race and Institutions.—There are many excellent reviews on the subject of the freedom from or susceptibility to decay amongst different races (Pickerill,[51] Hess,[18] Menzies, Campbell, Waugh[42]). Many theories have been evolved to explain the results. If attention is given to the presence or absence of vitamins, fresh fruits, milk (raw or boiled), coarse vegetables, meat, the quantities of calcium or phosphorus, or the amount of sunshine, there is endless confusion; but if, as Pickerill has admirably done, we focus our attention on the quantity of readily fermentable carbohydrate, then there is found in practically every case a direct relationship between caries and diet. Caries is rampant where the consumption of sugar and soft starches is high; it is absent when starches are not eaten. Evidence from institutions supports the same thesis (McBeath,[30] Sprawson[39] and Hess[18]). In institutions and orphanages the diet has frequently been found to be poor in milk, fresh fruits, green vegetables, calcium, phosphorus, vitamins, either severally or together, and there is generally an absence of dental hygiene, yet caries is infrequent. In all these cases there has been a low intake of readily fermentable carbohydrate.

Changes in Dietary Habit.—There is a general consensus of opinion that the incidence of caries has steadily increased, and this in spite of a steady decline in general morbidity, an increased attention to oral hygiene, and an increased use of vitamins and food rich in calcium and phosphorus. In short, if those factors which are considered of paramount importance by the antagonists of the bacterial theory were of any moment, dental caries should rather be decreasing. The progressive deterioration of the teeth, however, can be correlated with a change in dietary habits that has acted in a positive sense, and this change has been a progressive increase in the consumption of sugar and soft starches. The consumption of imported sugar in England has increased from three pounds per head at the beginning of the nineteenth century to 101·3 pounds per head in 1928-1929, and in the United States of America from 50 pounds per head in 1885 to 108·1 pounds per head in 1929.

Waugh has clearly shown that dental caries and the increased consumption of fermentable carbohydrates have gone hand in hand amongst the Eskimos. One thousand consecutive shops in the main shopping centres of Sydney were counted; of these, 40% sold sweets and easily fermentable carbohydrates. There is no shortage of pabulum, and with the possibility of 1,800,000 acidophilus bacilli to the cubic centimetre of saliva, there is adequate inoculum.

Considerations for the Diagnosis and Control of Caries.

There is almost conclusive evidence that dental caries is due to the solution of the enamel of the tooth by lactic acid produced in areas protected from the saliva. This acid results from the fermentation by *Bacillus acidophilus* of sugars and soft starches. Hadley,[15] of the Bunting research group, has provided a practical method for determining on a quantitative basis the prevalence of *Bacillus acidophilus* in any case of caries and has shown that in the absence of a suitable pabulum the number of organisms decreases enormously. It is now practicable for those interested in the ætiology of caries to obtain personal evidence for or against the bacterial theory of dental caries and to follow the effect of changes in diet.

One can anticipate that no effort on the part of either the medical or dental profession will determine a substantial decrease in the consumption of sugar or readily fermentable carbohydrate, but much might be done by educating parents in the feeding of children, the two important considerations being to give no sweets between meals and to give raw fruit at the end of each meal. Attention should also be given to devise improved methods of removing food *débris* from between the teeth.

Acknowledgement.

We are indebted to Dr. Haggett for assistance in the histology of the tooth and for other help.

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Reports of Cases.

ANTIPERISTALSIS IN THE URETER: A CASE SHOWING FILLING OF THE KIDNEY PELVIS FROM THE FILLED BLADDER.

By J. Leon Jona, D.Sc. (Adelaide), M.D. (Melbourne), M.S. (Adelaide), F.R.A.C.S.,
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In a recent communication[1] on the physiology and pathology of the kidney pelvis the author has drawn attention to the frequency with which antiperistalsis in the ureter is seen when the filled kidney pelvis is observed under the fluorescent screen. It was pointed out that this is especially seen when the urinary bladder is full. The following case illustrates the condition.

Clinical History.

The patient has had cystitis for years, with frequency of micturition day and night, and when only about two ounces of water were run into the bladder the patient complained of pain in the bladder. She had suffered from dorsal backache for years and pain in the left groin.

A radiogram showed a stone in the left ureter near the brim of the bony pelvis.

A catheter was passed along the left ureter to the left kidney pelvis. On filling the left kidney pelvis the "Abrodil" was quickly passed down the left ureter into the bladder and in the radiogram (Figure I) is seen mounting up the right ureter. The two successive radiograms (Figures II and III) show the fluid mounting up the ureter, showing waves, and entering the right kidney pelvis. The ureteric orifice appears definitely closed in two of the pictures (Figures I and III) and partly open in Figure II.

Another examination was conducted three weeks later. In this case no ureteric catheter was passed, but twenty cubic centimetres of "Abrodil" were injected into the bladder with a syringe. Under the fluorescent screen small globules about 1·9 centimetres (three-quarters of an inch) long were seen running in a stream up the left ureter. Occasionally this stream stopped and a normal wave passed down the ureter and several globules were evacuated into the bladder. On the left side a section of the ureter, about 5·0 centimetres (two inches) long, became filled from the bladder, and this long "globule" was passed up the ureter. The two pyelograms (Figures IV and V) demonstrate both these points and also the entry of the "Abrodil" into the pelvis of each kidney.

Comment.

From the author's experience of several hundred pyeloscopies it appears to him that antiperistalsis along whole or part of the ureter is relatively a common condition in the cases investigated. As stated above, this is especially seen when the bladder is full. Whether the condition exists in the normal individual in these cases has not been determined, but it certainly has been seen in the apparently normal case, when it did not appear until the kidney pelvis was apparently filled to capacity. It is suggested by the author that Nature has here a physiological method of limiting the secretion of urine in cases where the outflow of urine *per urethram* is obstructed (voluntarily or involuntarily) or where the kidney pelvis is unable to empty itself owing to spasm or obstruction somewhere along the course of the ureter. The waves of antiperistalsis carry the urine back into the kidney pelvis and possibly back into the calyces, producing a rise in pressure in the urine in the collecting tubules up to the secretion pressure of urine in the essential parts of the kidney substance with consequent inhibition of secretion till the resistance to outflow is removed. This damming back of the urine in the kidneys is always accompanied by kidney pain.

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Reviews.

THE PITUITARY BODY AND THE HYPOTHALAMUS.

THE idea of collecting and publishing in one volume four papers by Harvey Cushing on the pituitary body and correlated topics under the title of "The Pituitary Body, Hypothalamus and Parasympathetic Nervous System" was indeed a happy one.[1] This volume certainly meets the convenience of those already interested in the subjects discussed, as the author suggests it may, but we feel that it will do much more than this and that it will awaken interest in the well protected and still mystic area of the anatomically insignificant 'tween brain in anyone who will but open its covers.

The first paper included was the basis of the Lister Lecture originally published in *The Lancet* of July 19, 1930. In this the author discusses primitive views of the pituita, the birth of the concept, internal secretion, and of the new subject of endocrinology, adenomata of the glands with internal secretions, and particularly the three adenomata of the pituitary. He emphasizes the differences between their constitutional manifestations and their so-called "neighbourhood symptoms", which, as he stresses, more frequently lead to surgical intervention.

The relationship between the basophile elements of the anterior hypophysis and sexual dystrophy is discussed and the importance of pressure within the sella is demonstrated. Attention is called to the symptoms peculiarly associated with the craniopharyngiomata, that is, polyuria, adiposity, impaired growth, and sexual dystrophy. The hæmodynamic, the antidiuretic, the oxytocic, the pigmentary, the galactogue, the antipyretic, and the sudorific effects of lesions of the diencephalon-hypophyseal mechanism are dealt with and the close physiological inter-relationship which exists between these anatomically related structures is reviewed.

The second article, reprinted from the *Proceedings of the National Academy of Sciences*, bears the title "Posterior Pituitary Hormone and the Parasympathetic Nervous System". This paper records some personal investigations of the author on the effect of intraventricular injection of pituitrin, pilocarpine and atropine, and draws attention to the important rôle in neurophysiology played by the posterior lobe of the pituitary. As a result of these investigations the author asserts that in the diencephalon there is a parasympathetic head station and that normally pituitrin is secreted into the cerebro-spinal fluid as the hyaline bodies of Herring and by diffusion through the ependymal lining of the ventricular wall reaches the anterior nuclei of the hypothalmus on which it exerts its effect. When injected into the ventricles, pituitrin and pilocarpine cause a reaction which is characterized by widespread vasodilatation, by sweating, by retching and vomting, and by a marked lowering of the body temperature, and by a coincidal lowering of the basal metabolic rate. Atropine when given either subcutaneously or intravenously along with either pilocarpine or pituitrin given into the ventricles, is able completely to counteract this effect, as also is "Avertin", which presumably causes its effects by action on the diencephalic nuclei. The characteristic effect of pituitrin when injected into the ventricles is not produced if an insufficient dose of the drug is employed or if the ventricles are grossly dilated, or if the hypothalmic nuclei are destroyed by tumour. The author finally concludes that the neurohypophysis may conceivably bear a somewhat similar relation to the parasympathetic division that the medulla of the adrenal gland does to the sympathetic division of the vegetative nervous system.

The author, in the third paper, which is reprinted from the *Bulletin of the Johns Hopkins Hospital*, discusses pituitary basophilism and retraces the path he trod before he finally recognized the syndrome associated with a basophile adenoma of the anterior lobe of the pituitary. So far the author has been able to collect the histories of sixteen cases. He thus briefly describes the syndrome: (i) A rapidly acquired, peculiarly disposed, and usually painful adiposity confined to the face, neck and trunk, the extremities being spared; (ii) a tendency to become round shouldered (kyphotic) even to the point of measurable loss of height, associated with lumbo-spinal pains; (iii) a sexual dystrophy shown by early amenorrhœa in the female and ultimate functional impotence in the male; (iv) an alteration in the normal hirsuties, shown by a tendency to hypertrichosis of face and trunk in all females as well as in preadolescent males and possibly the reverse in adult males; (v) a dusky or plethoric appearance of the skin with purplish *linæ atrophicæ* particularly marked in the abdomen; (vi) vascular hypertension; (vii) a tendency to erythema; (viii) variable backaches, abdominal pains, fatigability and ultimate extreme weakness. He reports one case of a patient suffering with this syndrome who appears to have been benefited by X ray treatment.

"Peptic Ulcer and the Interbrain" is the title which the fourth paper bears. It was the basis of the Donald Balfour Lecture given at the university at Toronto and was originally published in *Surgery, Gynecology and Obstetrics*. Three cases, in which the patients, following operation on the cerebellum by the suboccipital approach, died of acute perforative lesions of the upper alimentary tract, are reported in detail. *Ante mortem* erosions of the gastric mucosa, gastric erosions and perforations accompanying malignant hypertension, gastromalacia, are discussed and the literature on them is reviewed. The theories of the pathogenesis of peptic ulceration are critically analysed and the author concludes that the consideration of the neurogenic aspect of ulcer pathogenesis along with certain experimental observations leads one to the opinion that in "highly strung persons, who incline to the form of nervous instability classified as parasympathetic, through emotion or repressed emotion incidental to continued worry and anxiety and heavy responsibility combined with other factors such as irregular meals and excessive use of tobacco, are particularly prone to have chronic digestive disturbances with hyperacidity often leading to ulcer".

The book is well printed in attractive type on art paper and worthily presents another milestone in the uncovering, by the distinguished author, of the secrets of the pituitary body.

[1] "Papers Relating to the Pituitary Body, Hypothalamus and Parasympathetic Nervous System", by H. Cushing; 1933. London: Baillière, Tindall and Cox. Super Royal 8vo., pp. 242, with 99 illustrations. Price: 29s. net.

FRACTURES.

Of all the books on fractures that we have seen in recent years, we think that the latest, a book by Dr. P. B. Magnuson, although only of moderate size, is one of the best.[1] We can say that there is none better. The author tells us in his preface that "all the methods described in this book have been thoroughly tried in practice. There are many more which are as good, there may be some that are better, but these have worked." The reader who is himself a practical man discovers very soon that Dr. Magnuson is another practical man, writing about what he has tried and found to be useful. Naturally these methods will not be in every case the favourite methods of the critic who has his own fund of experience, but they will be considered with interest and becoming respect. The younger practitioner who is looking for help and guidance will get it, and will not be left in a whirl of doubt as to which of several things he ought to do.

[1] "Fractures", by P. B. Magnuson, M.D.; 1933. Philadelphia: J. B. Lippincott Company; Australia: Angus and Robertson. Royal 8vo., pp. 466, with 317 illustrations. Price: 37s. 6d. net.

The author uses the Thomas splint, more or less modified, for many fractures of the lower extremity. He uses some splints and some methods that we have perhaps tried and discarded, some that we have never tried. He is acquainted with Böhler's fracture technique, but takes his own line. The treatment of the fractured carpal navicular bone is a case in point. He recognizes the difficulties in the treatment of fracture of the calcaneus, and the cutting of the Achilles tendon that he recommends is a good way of meeting a part of the difficulty.

At every step Dr. Magnuson discusses the injury in the light of claims for workers' compensation. This feature is welcome and it is well done. He has evidently had experience in that side of fracture work.

While the whole book runs to 466 pages, 50 pages are taken up by the discussion of spinal and skull fractures, a subject taken by Loyal Davis. There is a further chapter by J. S. Coulter on physical therapy in the treatment of fractures. They are both good, the chapter by Davis being excellent.

The book is well and usefully illustrated.

FOREIGN BODIES IN THE RESPIRATORY AND ALIMENTARY TRACTS.

"Foreign Bodies in the Air and Food Passages" is a valuable work based on a clinical experience of three thousand cases seen by Chevalier Jackson and Chevalier L. Jackson at their famous clinic in Philadelphia.[1] The book, though of greater interest to the specialist, should be of value to the medical profession generally.

A convenient arrangement for ready reference is the numbering of the paragraphs in the various chapters. The first chapter deals with the discovery and localization of the foreign body; and to be thoroughly carried out this examination must include the head, neck and trunk.

The authors recommend a preliminary fluoroscopy, but skiagrams must be made in all cases, as it is so easy to miss small objects when the screen is being used. The lateral view is as important as the antero-posterior, and by a study of the two films a fair idea can be gained of the particular bronchus involved.

Cervical deformities may cause trouble in introducing the endoscope and should be reported upon by the radiologist, while it is also important to exclude thymic enlargement in children.

Fluoroscopy is valuable in cases of non-opaque foreign bodies, as it is possible to determine differences in aeration due to bronchial obstruction. During fluoroscopy also an "asthmatoid" wheeze may be heard at the open mouth, and this is considered diagnostic of a foreign body in a bronchus. Another point of importance is stressed by the authors, namely, that the lower lobes are really posterior lobes, that the right middle lobe is an anterior one, and the upper lobes are anterior.

It is advised that no opaque oils or mixtures be used before skiagrams have been taken.

Foreign bodies in the œsophagus generally lie in the coronal plane, while in the larynx and trachea they lie in the sagittal plane. In the case of non-opaque foreign bodies in the œsophagus, a capsule of bismuth subnitrate is given, and if this is held up and does not pass after the patient takes a drink of water, or if it disintegrates, the diagnosis of foreign body is justified.

Radio-transparent foreign bodies in the lung cause difficulty in diagnosis, and this depends chiefly on differences of aeration due to valvular obstruction of a bronchus and later pathological changes.

Obstructive emphysema and atelectasis are best noted on the screen. In emphysema the heart shadow moves sideways on expiration and returns to its normal position on inspiration, whereas in atelectasis the heart remains still.

Foreign bodies in the stomach and intestines offer difficult problems. If the foreign body can pass the pylorus and is not likely to cause damage to the intestines, it should be left alone. If it passes into the intestine and remains at the same site for four or five days, a laparotomy should be done. The authors state that most gastric foreign bodies can be removed with a gastroscope.

The old practice of inverting patients who have inhaled or swallowed foreign bodies is roundly condemned.

There is an interesting chapter on the fluoroscopic control of the endoscope by using two screens at right angles. This is extremely difficult and requires much practice. The relation of the end of the tube to the foreign body is established and the tube is advanced towards it by picking the correct bronchus by sight and gently advancing the endoscope.

A series of excellent skiagrams illustrate practically every class of foreign body likely to be inhaled or swallowed, and the description beneath each plate is clear and concise.

The book is marred in parts by the use of manufactured words, such as "vegetal" for "vegetable", and "œsophageal pinchcock" for cardiac sphincter.

[1] "Annals of Roentgenology: A Series of Monographic Atlases", edited by J. T. Case; Volume XVI: Foreign Body in Air and Food Passages, by Chevalier Jackson, M.D., and Chevalier L. Jackson, M.D.; 1934. New York: Paul B. Hoeber. Demy 4to., pp. 285. Price: $12.00 net.

HÆMATOLOGY.

"Blood Diseases in General Practice", by A. Piney, is one volume of a series of pocket monographs on practical medicine.[1] It presents a brief account of modern hæmatology, with special reference to diagnosis and treatment. The reader is presumed to have some knowledge of the various types of blood cells.

The anæmias of infancy and childhood and of the adult life are reviewed in separate chapters. The following extracts show the practical nature of the work. A dirty tongue, the presence of free hydrochloric acid in the gastric juice or failure to elicit a satisfactory reticulocytosis within a week of starting adequate liver therapy will suggest revision of the diagnosis of pernicious anæmia. For languor persisting after the blood picture of a patient with this disease has returned to normal, a short course of Fowler's solution by mouth is said to be almost a specific. "Depot" injections of liver extract intramuscularly are advocated if treatment by mouth proves irksome. Larger doses of liver extract or desiccated stomach than are necessary to maintain a normal blood picture are recommended in an attempt to prevent the onset of subacute combined degeneration of the cord. A maintenance dose of iron is advised to prevent relapse in the hypochromic idiopathic anæmia of middle-aged women. The dysphagia of the Plummer-Vinson syndrome may not be relieved by cure of the anæmia with iron, while carcinoma of the pharyngo-œsophageal junction may supervene.

The allegedly simple classification of purpuric diseases is probably too complicated to appeal to the majority of practitioners, most of whom would also wisely prefer to avoid the risks associated with splenic puncture, which is suggested as a diagnostic aid in the aleuchæmic stages of chronic myeloid leuchæmia. In the treatment of *polycythæmia vera*, injections of phenylhydrazine hydrochloride are suggested, but no mention is made of its administration by mouth nor of its use by a weekly maintenance dose.

Short chapters on Hodgkin's disease, some diseases of the spleen, and glandular fever and agranulocytosis complete an admirable review of the present practical knowledge of hæmatology which can be strongly recommended to students and general practitioners.

[1] "Pocket Monographs on Practical Medicine: Blood Diseases in General Practice", by A. Piney, M.D., M.R.C.P.; 1934. London: John Bale, Sons and Danielsson. Foolscap 8vo., pp. 92. Price: 2s. 6d. net.

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THE SELENIDE TREATMENT OF CANCER.

Very little has been heard during the last two or three years of the treatment of cancer by colloidal suspensions of lead. This method of treatment, which was introduced by the Liverpool workers headed by Blair Bell, sometimes gave surprising results, especially in the later stages of cancer. Many people hoped that work on the lead treatment of cancer, together with investigations in radiation therapy, might be of some help in the discovery of the cause of cancer, but so far they have been disappointed. One worker whose interest in lead treatment has not flagged is A. T. Todd, of Bristol, England. He commenced research on the treatment of cancer in 1926, and in 1930 published in *The Lancet* the results of his work. His reports were characterized by frankness, modesty and an absence of unjustified claims. He has done most of his work with a combination of lead and selenium. Colloidal selenium alone is a very toxic substance; five cubic centimetres of a 0·1% suspension of selenium is sufficient to kill an animal of about two kilograms weight, but combined with lead as selenide, selenium is comparatively innocuous. In 1930 Todd explained that he gave lead selenide to increase the local and general defence mechanisms. He also insisted that other measures should be adopted at the same time. He gave calcium chloride to increase ionized calcium by acidosis, to correct alkalosis and to furnish a depôt for calcium assimilation. He gave fat-soluble vitamin in large doses in order to increase calcium absorption and to combat any infective element. He also gave small doses of thyreoid extract as a tonic and an anti-infective agent, and liver extract to combat anæmia.

Todd has made another report on his work with lead selenide.[1] In bringing this report to the notice of readers, it will be possible to give merely an outline of what has been done; all those engaged in the treatment of cancer will, it is hoped, study the report in detail. It must be explained that all the patients treated by Todd have been in the late stages of the disease; they have been the rejects of surgeons and radiotherapists. In many instances the disease has been so advanced that patients have died before they could be admitted to one of his beds. For some years he used a selenide known as "D4S"; the colloid he is now using is "R.A.S." With "D4S" he was able to produce 10% of four-year cures, and in about 50% of cases life was prolonged, as a rule with comfort to the patients. Todd now combines the use of "R.A.S." with radiation, and it is interesting to see how his method was evolved. He found that when radiation was applied to patients who were impregnated with lead selenide, there was always a rapid intensification of the growth of the neoplasm; patients whose condition had been stationary for months died within a few weeks of acute cancerous invasion. After considering possible explanations for these phenomena, Todd adopted the hypothesis that the presence of the selenide converted a relatively small into a relatively large dose of radiation. He found that by reducing selenium dioxide in the presence of sulphur colloid, selenium was taken up and a double colloid of selenium and sulphur resulted. This new colloid, known as "SSe", is now available in addition to "R.A.S." Todd assumes that cancer is an infective disease with a defence mechanism; if this is true, he states, growth would be inhibited with adequate stimulation of the defence. In sup-

[1] *The British Journal of Surgery*, April, 1934.

port of this view he points out that radiation alone, if correct dosage is employed, increases the amount of what he calls the junction tissue and of the macrophages. Lead selenide alone and lead selenide given after radiation give the same result, and with increase of junction tissue there is atrophy of neoplastic tissue. On the other hand, radiation of the same dosage after the administration of lead selenide causes a poverty of the junction tissue, usually with disappearance of the macrophages and an increased growth of the implant. In his newer method of treatment Todd uses radiation followed by the injection of selenium. He claims that some change follows the application of deep X and γ radiation to a tissue impregnated with selenium colloid; and he advances certain proof in support of his contention.

Todd gives the details of his method of treatment; since the details are not of importance in this discussion no further mention will be made of them. We are not concerned particularly with the numbers of the patients who have recovered, but we may point out that among one group of 72 cases there are 15 apparent cures; in a second group of those more recently treated, three patients are apparently cured, nine are doing well, four are doing badly, and twelve are dead. What we are concerned with may be stated under two headings. First of all, Todd has reawakened interest in the chemotherapy of cancer and has described a method that gives some hope to those who, according to accepted criteria, are beyond all possible aid. It is important to understand his insistence on the strictest attention to details in technique if his method is to be used, and it is also well to realize that the patients subjected to this treatment should be those whose mental calibre is such that they would be prepared to undergo the many months of treatment that are necessary.

The second aspect of this work is that dealing with future research. There are those who hold that effective research cannot be carried out by therapeutic methods. The way in which Todd discusses his work shows that such a statement is not well founded. The possibilities opened by work of this kind are enormous. Blair Bell was right when he insisted that a working hypothesis should be explored in every possible direction by cooperation of all parts of the "aggregate mind". He deprecated the examination of a number of ideas in one direction only according to the special knowledge of the person interested. Workers in Australia are investigating the effects of radiation on the cancer cell. If the method of treatment used by A. T. Todd were adopted in Australia with the meticulous attention to detail that appears to be necessary, not only would hopeless sufferers be likely to receive some benefit, but cancer investigators (radiologists, physicists, chemists and pathologists) would have a further and most interesting field to explore.

Current Comment.

NUTRITION AND ARTHRITIS.

Without entering upon the more provocative elements in the causation of arthritis, it will be agreed that it is a disease which both influences nutrition and is profoundly modified by changes in nutrition. When pain and impaired mobility affect activity it is evident that the general metabolic well-being of the body must suffer and also that of the tissues of the joints. But it is also important to consider the part played by errors in the digestion and absorption of foodstuffs in the inception and maintenance of joint changes. Whatever may be the effect of open or cryptic infection, resistance must be a supremely important factor, and this in its turn depends largely upon conditions of life and environment, and to a certain degree also upon food. The endocrine factors are no doubt important in addition in some cases, but whereas these may not be easy to modify, and while it may, for economic reasons, be difficult to transplant patients to other and more favourable environments, it is usually possible to amend their diet and perhaps to assist their capacity to absorb food. This is the text of an article by F. L. Burnett and F. R. Ober, in which they make great claims for treatment directed towards the establishment of anabolic nutrition.[1] They maintain that the rate of intestinal movement is an index of absorption, and as this in turn will depend upon the correct functioning of the proximal part of the colon, the process of digestion, with subsequent assimilation, is more likely to be normal if care be taken that the colonic activities are not unduly hurried. It seems reasonable to assume that a certain deliberateness of intestinal movement will favour correct nutrition and that the type of the dejections will indicate whether this ideal is being attained. Of course, it would be possible to push this argument too far, for constipation would hardly be regarded as an

[1] *The American Journal of the Medical Sciences*, July, 1934.

alimentary ideal. But these authors have made some observations on the digestive faults found in the subjects of arthritis. The commonest fault lay in the digestion of fat; starch digestion was also frequently found to be defective and, least common of all was defective digestion of protein. It has been previously pointed out in these pages that in spite of the not uncommon defects in gastric secretion, notably lack of hydrochloric acid, in many persons (particularly the sufferers from arthritis), protein digestion is usually well accomplished. It is also well known that excess of carbohydrate is not advisable in the dietary of arthritics, and these findings are thereby confirmed. But the point of the contentions of Burnett and Ober is that patients should not only be given instructions as to diet, but that they should be taught to observe the nature of the dejections and trained so to regulate their food intake and their habits that they produce firm and segmented fæces. If intestinal hurry is thus eliminated, and if a correct and non-irritating diet is employed, it is claimed that great clinical improvement is effected in these patients. They are given printed outlines to fill in, and are expected to keep the necessary records of their digestive and excretory activities. Lest it should be thought that the authors rely on these methods only, it should be stated that they advise rest, heat, massage, and other forms of physical therapy under skilled direction. They also stress the importance of the correct use of rest and exercise. However, the perusal of the article reveals that the chief article of the authors' faith is in the adjustment of the function of the proximal part of the colon. Remembering the commonness of some aberrations of the digestive tract in arthritis, such as achlorhydria and redundancy of the colon, it cannot be denied that more attention might be paid to the dietetic treatment of this disease. Perhaps all will not follow these authors entirely in their enthusiasms, nor would there be universal agreement as to the desirability of making an already rather self-conscious public even more acutely aware of its more intimate functions. Nevertheless it is well to be receptive of ideas, for there are still instances found in which the sole dietetic advice given to arthritis patients has been that false and unnecessary injunction that they should abstain from red meats.

INTESTINAL AMŒBIASIS.

The treatment of intestinal amœbiasis of long standing is often very difficult. Many people seem to think that the only requirement is a series of injections of emetine. This is far from correct. Emetine is of incalculable value in the treatment of acute amœbic dysentery; but it seldom suffices to eradicate the infection. Indeed, in some cases no treatment seems to have any lasting beneficial effect. Occasionally one recognized method of treatment seems to succeed where numerous others have failed; in such cases the possible influence of the patient's resistance should be borne in mind. A great deal of work has been done in India during the past few years by H. W. Acton, R. N. Chopra and others. Among other drugs, Acton and Chopra have investigated certain alkaloids of *Holarrhena antidysenterica* (known locally as *kurchi*). They have shown, for example, that kurchi bismuth iodide is often of great value in the treatment of chronic amœbiasis; but they find difficulty in obtaining an adequate supply of raw material of uniformly good quality for its manufacture. Chopra and B. and S. Sen last year investigated an organic iodine preparation, a proprietary article known as "Carbarsone"; more recently Chopra and S. Sen have reported the results of a further trial of this drug.[1]

"Carbarsone" is given by mouth in gelatine capsules in a dose of 0·25 gramme twice a day for ten days. If at the end of this time *Entamœba histolytica* can still be found in the stools, a further course lasting for five days is given. The only additional treatment consists in the administration each day of a saline purgative. If the infection persists, Chopra and Sen advise the administration of kurchi extract by mouth for several months, when "Carbarsone" can be given again if necessary. They prefer not to repeat the "Carbarsone" treatment earlier, on account of the possibility of arsenical poisoning. "Carbarsone" effected an apparent cure in four of a series of five cases of acute amœbic dysentery; in the fifth the patient died of pneumonia before the investigations could be completed. Of seventeen patients with chronic amœbiasis, thirteen were apparently cured and two were still passing *Entamœba histolytica* in the stools at the end of ten days' treatment; two others left hospital before the completion of the investigations, but were apparently not passing *Entamœba histolytica* in the stools at the time.

Chopra and Sen have treated fifty-three patients in all with "Carbarsone", and have reached the conclusion that "it is likely to prove a very valuable drug in the treatment of intestinal amœbiasis". Advantages are: that it can be administered by the mouth, it promptly relieves clinical symptoms, it is suitable for administration to out-patients, it apparently has no toxic effects in the doses employed, and it "exercises a remarkable tonic effect on the system in general". They point out further that the drug appears to be equally efficacious in acute and chronic amœbiasis, the cure rate being roughly the same in both.

Other arsenical preparations are employed in the treatment of intestinal amœbiasis, and some of them have been the subject of enthusiastic reports. They have their value; but it is doubtful whether they can be regarded as capable of eradicating amœbic infection. The wise physician will not hasten to give "Carbarsone" with unrestricted freedom; but he will note that Chopra and Sen have carried out their work with care and have accepted only the strictest criteria of cure.

[1] *The Indian Medical Gazette*, July, 1934.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Ovarian Substances in Hæmophilia.

R. P. Stetson *et alii* (*The Journal of the American Medical Association*, April 7, 1934) report failure to produce effect in prolonged use of various ovarian substances in severe cases of hæmophilia. Large amounts of one or another of eight different preparations were given over periods of from twenty-eight to eighty-one days. In no instance was the coagulation time of the venous blood found to be depressed as the result of such therapy. Theelin administered subcutaneously to two patients for from three to seven days neither stopped bleeding nor reduced the clotting time. No clinical improvement was noted which could be attributed to ovarian therapy. In four of the patients hæmarthroses developed late in the course of treatment without obvious trauma or infection.

The Effect of Chloral Hydrate on the Maternal and Fœtal Organisms.

R. E. Campbell (*American Journal of Obstetrics and Gynecology*, July, 1934) records his investigations on the action of chloral hydrate on the liver, heart and kidneys of pregnant dogs. The method was to remove a portion of the liver under ether anæsthesia and at the same time one of the pups. Then in a few days' time chloral hydrate was administered. After delivery the organs of the dog and pup were examined and compared with the tissue previously removed. The author concludes that chloral hydrate has a definite pathological effect on the liver, heart and kidneys and has no place in the medical treatment of the toxæmias of pregnancy.

Erysipelas.

B. Fantus (*The Journal of the American Medical Association*, July 21, 1934) discusses the treatment of erysipelas. Isolation from surgical patients is desirable. Disinfection of the hands of nurses and physicians is essential. Ultra-violet irradiation of the area affected and for 5·0 centimetres (two inches) beyond is indicated. This is carried out by means of a mercury quartz burner for ten minutes at a distance of 30·0 centimetres (twelve inches); usually one intense treatment suffices. Röntgen rays, 100 kilovolts, unfiltered, and not over one-fourth of an erythema dose, should be applied and repeated once on the second day, except in the diabetic, in the nephritic and in young children. Cold evaporating lotions with 1% phenol added relieve the hot, burning sensation; aluminium acetate diluted 1 in 8 is preferable for moist areas. Erysipelas antiserum should be preceded by an intracutaneous test, 0·1 cubic centimetre of the serum diluted 1 in 10 being used; if no result occurs, one may inject the remainder of the contents of the ampoule. The dose may be repeated daily for three days. A hypnotic may be required for obstinate insomnia or for stimulation for collapse.

The Use of Bismuth Subnitrate in Arterial Hypertension.

Curtis Bruen (*The American Journal of the Medical Sciences*, July, 1934) refers to the findings of Stieglitz, who claimed to have obtained beneficial results in the treatment of arterial hypertension by giving bismuth subnitrate in doses of 0·6 gramme (ten grains) three times a day. Stieglitz reported that the average systolic and diastolic blood pressures reverted toward the normal maximum level. On the other hand, Ayman, in a small series in which allowance was made for control factors, found that bismuth subnitrate in such dosage was without demonstrable effect on the blood pressure. The author has made an investigation in twenty cases. The ages of the patients ranged from twenty-seven to seventy-three years, with an average of fifty-one. The stated duration of hypertension ranged from a few months to nineteen years, with an average of three years. The author found that the variations in blood pressure could be invariably attributed to adventitious factors. He gives details of the controls adopted in his investigation and claims that bismuth subnitrate, when given by mouth, even in the largest therapeutically practicable doses, does not develop sufficient nitrite action to exert any demonstrable effect on the blood pressure or on the symptoms of arterial hypertension.

Intravenous Lethal Doses of "Amytal" in Animals.

H. G. O. Holck and M. A. Kanân (*The Journal of Laboratory and Clinical Medicine*, August, 1934) have investigated the intravenous lethal doses of "Amytal" in the dog and the rabbit, and give details of their findings. They have compiled a table of animal doses from the literature. The authors discuss the relative merits of the various factors which may modify "Amytal" dosage and refer to the desirability of clarifying the toxicology terminology by the adoption of the median lethal dose as a common criterion of fatality. They suggest that some authoritative body should suggest one or two criteria for each species of animal by which the minimum effective dose for new hypnotics could be established.

The Treatment of Anæmia in Children.

M. C. Lottrup (*American Journal of Diseases of Children*, January, 1934) has endeavoured to compare the effects of ferrous, ferric salts and reduced iron in the anæmia of children. He found that ferric salts caused a quick and considerable rise in the hæmoglobin percentage, while ferric preparations appeared to be without effect. Reduced iron was effective in certain instances. A carefully controlled series of observations showed cupric sulphate to be without effect, either alone or in combination with salts of iron. Unlike reduced iron, no dyspeptic symptoms were observed after the use of ferrous compounds. On these grounds, ferrous preparations are preferable for the treatment of simple (nutritional) anæmia.

Gold Tribromide in Pertussis and Other Spasmodic Disorders.

J. Epstein (*Archives of Pediatrics*, January, 1934) has classified the bromides used therapeutically into monobromides, dibromides and tribromides, and has decided that the tribromides are the most efficacious group of sedatives. As an elixir, gold tribromide has, in the author's hands, proved most efficacious in pertussis and a number of other spasmodic disorders, such as bronchial asthma, chorea, migraine and *petit mal*. He states that gold tribromide also gives decided relief in spasmophilic disorders. He thinks that the antibacterial effect of the gold ion indicates its value in infections of the respiratory system, as in pulmonary tuberculosis.

Alcohol Injection of the Sympathetic Rami in Bronchial Asthma.

G. L. L. Levin (*The Lancet*, August 4, 1934) points out that the bronchial rami contained in the sympathetic dorsal fibres are comparatively superficial at one part of their long course, namely, at the point where they join the corresponding intercostal nerves. At this point they are open to attack either by the knife or by injections of absolute alcohol. The author has treated sixteen patients by injections of absolute alcohol. He gives details of his technique. Most of the patients were thought to be incurable. In ten instances complete relief occurred after four to eight injections. In one case there was sufficient improvement after two injections for the patient to discontinue treatment. Three patients improved considerably after two or three injections. In two cases the improvement was only slight. The author thinks that the patients best suited to this type of treatment are those whose asthma is of long standing and inveterate.

NEUROLOGY AND PSYCHIATRY.

Cerebro-Spinal Fluid Changes in Multiple Sclerosis.

H. Houston Merritt (*Brain*, Volume LVII, Part 1, 1934) reports upon 152 investigations from 100 cases of multiple sclerosis. All cases in which the diagnosis was in doubt were excluded from the survey, and in every case the Wassermann test upon both blood and cerebro-spinal fluid gave no reaction.

Together with 152 investigations of his own, this author reviews the findings of a further 968 previously reported in the literature, from which he concludes that the cerebro-spinal fluid is absolutely normal in less than 20% of cases of multiple sclerosis; that the spinal fluid pressure is usually within normal limits; that there is a moderate increase in the number of cells; that the protein content of the fluid was increased in 45% of the reported cases. An abnormal colloidal gold curve was present in 71% of cases. There are no changes in the cerebro-spinal fluid that can be regarded as pathognomonic of multiple sclerosis; but the presence of an abnormal colloidal gold curve with a slight or moderate pleocytosis and an increased protein content is highly suggestive of multiple sclerosis, particularly if the possibility of a syphilitic condition can be excluded.

The Blood Sugar in Relation to Emotional Reactions.

JOHN C. WHITEHORN (*The American Journal of Psychiatry*, March, 1934) reports on a group of 958 mental patients observed over a period of twelve years, as the result of which he concludes that, contrary to an accepted belief, hyperglycæmia has little connexion with emotion. Indeed, he believes that as a general rule psychotic patients showed more uniformity within the normal blood sugar range than the "clinically normal". Of 151 manic-depressive patients only one showed hyperglycæmia associated with affective disturbance. Of the 107 patients in this series classified as suffering from *dementia præcox*, two only showed hyperglycæmia. This observer further notes that hyperglycæmia is very uncommon in young patients, whatever their emotional states, while it tends to be commoner in elderly patients with organic psychoses. Melancholic reactions appear to depress somewhat the insulinogenic function of the pancreas.

Lipoid Metabolism in Mental Disorders.

J. S. SHARPE (*The Journal of Mental Science*, January, 1934) reports upon an investigation of lipoid metabolism in relation to psychotic states, particularly upon the so-called biogenetic psychoses, mania, melancholia and *dementia præcox*. He describes the technique of the choline test on the isolated frog heart. From a determination of the oxygen content of the venous and arterial blood a coefficient of utilization is calculated. This coefficient of utilization is found to be very low in these psychotic states. The author finds no important variation from the normal in the lecithin content of the blood in mental disorder. The cholesterol content of the blood is decreased by approximately 25% in the early cases of *dementia præcox*; but as chronicity of this disorder advances there is an appreciable increase in cholesterol, which the author considers may be due to the hyperactivity of the suprarenals. In melancholia and confusional insanity the cholesterol figure is on the high side, whereas in recurrent mania the blood cholesterol reading is particularly high, especially during the acute phase. Together with this, it has been observed that the coefficient of utilization is also very high, indicating increased metabolic rate.

Huntington's Chorea.

CLARENCE A. BUCK (*The Canadian Medical Association Journal*, August, 1934) records a family tree showing 16 cases of Huntington's chorea in a family history in which 32 persons came in the direct hereditary line. Although a number of those in the third generation, most of the fourth and all of the fifth generation have not yet reached the age group when the symptoms appear, this tree (the author points out) fairly closely follows the Mendelian theory of heredity. The author describes one case in detail. He draws attention to the hopeless prognosis and attacks the problem of treatment from the eugenic point of view—the only hope seems to be the extermination of the strain. "Huntington stock should not reproduce." In considering those who escape, the author believes that if both parents are free from the disease, their children will be free from it. Owing, however, to the fact that some of the lineage frequently die from other causes before they have reached the age at which symptoms appear, "we can only be dogmatic in saying that the third generation with grandparents and parents free of the disease will be assuredly free".

Myotonia Acquisita and Muscular Hypertrophies.

KNUD H. KRABBE (*Brain*, June, 1934) reports in detail a case of *myotonia acquisita*. He gives the histories of two other cases in which muscular hypertrophy was present. In all three cases the muscular hypertrophy took place after an acute illness. In the first case this was characterized by severe pains in the back, slight pain in the extremities and weakness of the limb muscles; subsequently a ready fatigue developed in the arms and hands. The second patient had a febrile illness and afterwards developed anisocoria, peripheral facial palsy and weakness in the legs. The third patient also had an acute illness characterized by pains and palsies of the legs, without fever. In all three cases, after the fever there occurred a true symmetrical hypertrophy of the muscles of the legs or of the arms. In one case (the first) the hypertrophy was associated with myotonic symptoms; in the two others no myotonia was observed. The author refers to the common statement that myotonia often presents a muscular hypertrophy as a special symptom. He suggests that muscular hypertrophy presents myotonia as a special symptom. He thinks that it may be supposed in all cases that the polyneuritis has caused a certain destruction of the muscles and that subsequently a compensatory hypertrophy has developed. The author discusses 34 cases of *myotonia acquisita* that he has found in the literature; he discusses these in some detail. He then summarizes the cases of true muscular hypertrophy described in the literature, twenty in number. He points out that true muscular hypertrophy generally develops after a polyneuritic syndrome or after a disease associated with polyneuritis. He draws two conclusions: (i) In most cases a polyneuritis recovers completely or it leaves a more or less pronounced atrophy of the affected muscles. (ii) In some cases the recovery is excessive and the muscles hypertrophy. Sometimes a muscle hypertrophies as a whole, presenting normal power or a slight weakness. In other cases the sarcoplasm especially hypertrophies, and in these a myotonic condition will be the consequence of the hypertrophy. The author adds that if these views are correct, *myotonia acquisita* and true muscular hypertrophy may be regarded as two variations of the same abnormality, an abnormal regeneration after neuritic processes.

Subacute Spino-Cerebellar Degeneration.

J. G. GREENFIELD (*Brain*, June, 1934) states that the spino-cerebellar ataxias are among those types of nervous disease that have a special tendency to vary from their main type. This tendency is more pronounced in the rarer cases of cerebellar ataxia which come on in the middle or later years of life. In the two patients described by the author, aged sixty-six and fifty-seven years, the condition ran a rapid course. Death occurred in three and in seven months respectively from the onset of the first symptoms. In both patients pains in the limbs were a prominent early symptom; this was succeeded by weakness and ataxia of the legs and arms, dysarthria and mental enfeeblement. Nystagmus was indefinite or quite absent and no loss of skin sensibility was found. There was considerable pleocytosis and excess of protein in the cerebro-spinal fluid in both cases. Pathologically the condition was characterized by degeneration of the long tracts of the cord, especially the dorsal columns and cerebellar tracts, with earlier degeneration of the pyramidal tracts. There was great loss of Purkinje cells in the cerebellar cortex, the granule cells being relatively spared. The superior cerebellar peduncles and the longer tracts in the tegmentum of the brain stem were involved in the second case. The nucleus of Luys and the strio-Luysial fibres were degenerated in both cases; this is stated by the author to have been the most striking new feature in the two cases. The *substantia nigra, nucleus ruber, nuclei pontis* and olivary nuclei were unaffected and the cerebral cortex was intact. In both cases there was fairly intense perivascular infiltration in the neighbourhood of the degenerated tracts.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association, in conjunction with the Australian Dental Association, was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on August 30, 1934, Dr. A. J. Collins, the President, in the chair.

Periodontal Lesions and Dental Caries.

Mr. T. T. Alkin read a paper entitled "Civilization's Food as the Possible Origin of Dental Disease" (see page 501).

Professor Harvey Sutton read a paper entitled "Notes on Periodontitis and Its Genesis" (see page 506).

Dr. F. S. Hansman read a paper by himself and Dr. F. Marshall, entitled "The Ætiology of Dental Caries" (see page 511).

Mr. R. Morse Withycombe said they had listened to three excellent papers, which, notwithstanding much valuable information, had missed the mark. Function alone controlled life, and structure was formed and maintained by it. An illustration of what this meant to dentistry might be exemplified by quoting a patient under observation. The life of a boy shortly after birth had been despaired of, the parents were delicate and both suffered from pyorrhœa and had very narrow and constricted dental arches. The child had been handed over to a specialist, who arranged the diet. The feature of most interest was that from two years of age the child was fed on dry, crisp food, any form of drink being strictly prohibited excepting between meals. The result was that not only was the boy forced to use his teeth, but to use them in the right way. Reverting to type, he had swung the mandible vigorously into lateral excursion and, quickly cutting facets on the occluding surfaces of the teeth, he had avoided the usual affliction of present day children—a locking of the teeth. He had developed under this powerful exercise wide-spreading dental arches which would assuredly make him immune to pyorrhœa and probably to caries. Dr. Harvey Sutton had mentioned that out of 100 boys examined, 100% had gingivitis. This serious symptom had developed in these boys owing to the teeth being in locked occlusion, causing a hopeless jarring of the teeth during mastication. The only means to save the coming generation was to insist on diet as mentioned for the infant, which would prevent the shortening of the transverse occlusal curve, and so the lower cuspids would find their way between the cusps of the upper cuspids and laterals.

Mr. Withycombe had examined 100 boys between twelve and fourteen years of age at a high school and found 99% with inflammatory symptoms of the gums. One fine lad alone had a most perfect set of teeth, but his teeth were well worn and worked like a machine. He never used a toothbrush, and his teeth were perfectly clean and gums free from any sign of inflammation.

Concerning the important question of the pyorrhœa pocket, Dr. Harvey Sutton had made some definite statements which the time limited prevented answering fully, but he would make some definite statements which would serve to reply in a measure to Dr. Harvey Sutton, as well as to Mr. Alkin. After treating over 4,000 cases of pyorrhœa, he was convinced that neither food—a dirty mouth—nor a lowered systemic resistance was a primary ætiological factor in the periodontal pocket. He was convinced that food had no primary ætiological influence on the disease, because he had not in any way tampered with the food of all the patients whom he had treated, and he had consistently obtained successful results, even with Vincent's disease, which was supposed to be a filth and food deficiency disease. Vincent's and such like contagious diseases were held at bay by perfect function. He did not consider a filthy mouth a primary ætiological factor, because he had treated so many cases by simply removing the calcareous deposits, and had found the gums perfectly healthy. Such patients, however, invariably had well worn facets to their teeth, showing that the function was good. He did not believe that a lowered systemic resistance was the primary ætiological factor, because he had seen so many people dying of chronic diseases whose gums were healthy when the occlusion was right. Even if they had only a few teeth left which had the occluding surfaces worn and in the same plane, there was never any sign of periodontal disease.

In conclusion, Mr. Withycombe stressed the importance of the ætiological factor as previously enlarged on; all other factors bore a superficial relationship to the disease. He had never seen a person with normal function of the teeth with symptoms of pyorrhœa. This one ætiological factor, embracing perfect function, was the only factor of the very many suggested in current dental literature which he had found could not be discarded without a recurrence of the disease.

Dr. J. W. Wilson said that the various schools of thought had been represented in the able discussion of the subject. There existed no cut and dried theory that could not be opposed. The present state of knowledge was too complex to be covered by any single line of approach.

In regard to the problem of periodontal lesions, lack of function undoubtedly played an important part in the ætiology; but if the body did not take in the necessary items for nutrition, then the cells could not maintain their normality.

There was some disagreement in regard to the caries problem. Overseas the chemico-parasitic relationship was considered of primary importance. The Mellanbys did not claim that diet alone was responsible for caries; all that they claimed was the susceptibility of the tooth if diet was inadequate. Clinically, however, hypoplasia often existed in spots and showed no susceptibility to bacterial invasion. They should aim at a diet that gave function and which at the same time contained adequate and balanced nutritional quantities. It was wrong to try to solve the problem in one direction only.

Mr. L. S. Beckett expressed his appreciation of the work that had been done on the subject. At the same time it was bewildering. What were they to tell their patients? The position was very confusing and he did not think that many would feel that they had the solution of either the pyorrhœa or the caries problem in a practical and usable form. Mr. Beckett said that there were about eighteen different theories of caries. The differences in the foods of races free from caries and pyorrhœa ranged from carbohydrate diets to meat diets—from one extreme type of diet to another. It seemed that immunity could exist on either type of diet, provided that those constituents still remained that would enable Nature to nourish and build the tissues in a normal way. There was too much fancy preparation of foods amongst civilized people. The diet of natives was simple and natural, and as a rule they did not try to digest more than one type of food at a time; certainly they did not indulge in seven-course meals.

Weston Price had observed that under natural conditions the teeth were found in excellent condition. Caries came with the use of white flour, sugar and tinned foods *et cetera*, and it attacked natives using these commodities in an even worse form than it attacked white men.

Dr. Hansman had said that the consumption of calcium, minerals and vitamins had increased under modern conditions. Mr. Beckett did not think this was so, as research reports in recent dental journals had analysed modern diets and had reported to the contrary. Weston Price maintained that the use of refined foods gave a higher fuel index with a less amount of food, and therefore a lower total mineral content. If people in sedentary occupations needed fewer calories in their diet and consumed refined foods, then there would of necessity be a dual reduction in the calcium and vitamins available.

It should be stressed that people in sedentary occupations must have a diet high in mineral content and vitamins, and these should even be artificially added to if necessary. Such people needed only a small amount of fuel, but it was necessary that this amount should

contain a larger proportion of mineral and vitamin building materials to keep the body in health; their food must therefore be carefully selected. The maintenance of the body cells, which were continually being rebuilt, made this necessary.

Mr. Beckett thought that Maddern's mechanical theory of caries was reasonable, if considered as one link in the chain. Under the enamel there was the elastic dentine. If this dentine was inadequately supplied with minerals, it was reasonable to suppose that it would more readily yield to stresses and crack the enamel surface. This would enable the onset of the caries to occur as set out in the chemico-parasitic theory. Some grain of truth could be found in many theories of caries, even in the discredited salt theory of Eckerman. Salt was often taken in larger quantities than necessary where there was a deficiency of minerals in the food, and thus was a symptom of mineral deficiency and associated with the presence of caries.

In conclusion, Mr. Beckett said that the volume of the literature was almost too extensive to keep track of. A great deal of classification, analysis and correlation was needed.

Mr. Owen S. B. Hogg said that no mention had been made of the connexion between faulty elimination and pyorrhœa. He spoke of the use of cellulose in the treatment of pyorrhœa and of vitamin *B* in the treatment of New Guinea mouth. Mouth lesions were certainly associated with systemic disturbances, and study along these channels should be profitable.

Dr. S. Lever supported Mr. Hogg's remarks. Many years' experience, he said, convinced him that faulty metabolism and elimination, often caused by incompatible foods as well as other diet factors, were the principal causes of both pyorrhœa and dental caries, owing to toxins affecting the soft tissues and the saliva by way of the circulation.

Dr. Lorimer Dods thanked the speakers for their papers. He had particularly appreciated Dr. Hansman's paper, which coincided with his own views on this problem. He would confine his remarks to dental caries in childhood, since this was the aspect of the subject of which he had had most experience. He had observed groups of children during their infancy at the Woolloomooloo Baby Health Centre and had been able to follow them up to four or five years of age at the Woolloomooloo Kindergarten. He had started with a cheerful and confident acceptance of the value of vitamins, sunlight, adequate calcium intake and restriction of carbohydrates. But he had gradually lost faith, for he had observed children, whose mothers appeared to have followed these desiderata both for themselves and their offspring, who exhibited gross dental caries, while some of those children who had received most unsatisfactory diets had excellent teeth. In his own practice Dr. Dods had tried to direct the diets of both mothers and infants along accepted lines, had ordered vitamins *A* and *D* concentrates and had emphasized the restriction of carbohydrates, and yet many of these children exhibited well marked dental caries.

In conclusion, Dr. Dods suggested that perhaps his findings were of little real value as evidence, and that possibly they reminded his audience of Whistler's statement about Oscar Wilde: "He had no enemies, but his friends disliked him very much!"

Dr. J. H. Halliday suggested that every child should see that it chose parents with a long line of good teeth. An hereditary factor must be present.

Dr. Rex Lane proposed to add to the confusion. He referred to the variability in susceptibility to caries of children of the same family. From his experience it seemed to Dr. Lane that all children with the same parents did not start off with an equal chance. Moreover, the susceptibility of each child varied from time to time. None of the theories so far advanced satisfactorily explained the various and apparently conflicting aspects of the problem.

Dr. A. J. Arnott said that it had been a very instructive evening. Periodontal diseases and caries presented problems for research workers as well as for general practitioners, both medical and dental. Professor Harvey Sutton had mentioned the work accomplished by Kirkpatrick and Clements in Vincent's infection and tropical ulcer; these two men had been associated in their work in Sydney during 1933 and had accomplished a great deal, and at present were working together in New Guinea. Dr. Arnott spoke of the dental research carried out at the University of Sydney and the Dental Hospital associated, and said that the Dental Research Laboratory was the most modern in the Southern Hemisphere. In the establishment of the Research Laboratory great assistance had been given by the School of Public Health and Tropical Medicine. Dr. Arnott suggested that on the return of the Dental Research Scholar and Dr. Clements from New Guinea, a combined meeting of the New South Wales Branch of the British Medical Association and of the Australian Dental Association should again be held, at which these two workers could tell members of the results of their research.

Dr. A. J. Collins, on behalf of the New South Wales Branch of the British Medical Association, extended his thanks to the Australian Dental Association for their cooperation at this meeting and for their liberal participation in the discussion. It had been an instructive evening. Dr. Collins thought that some of the apparent confusion was due to a failure to realize the difference in the problem of laying down tissues and later of maintaining them. Whereas adequate vitamin supply was important in laying down the tissues, their provision later was not effective in preventing the course of caries. In conclusion, Dr. Collins reechoed Dr. Arnott's wish for a further combined meeting in the future.

Mr. T. T. Alkin said that there was nothing to which to reply. He had come to the meeting as a humble student of the question and had in his paper put forward things that seemed important. He wished that some eminent medical man had come forward and told them what was the function of the glands controlling these things. Was it failure in function that prevented assimilation? In America and elsewhere they could produce experimentally the peculiar conditions found in periodontal disease. Surely such a condition must be due to some dietetic error, loss of function, or possibly both. Mr. Alkin was glad that he had not made definite statements from his own point of view, but contented himself by stating the results of research workers, hoping discussion would assist in better understanding of the problems.

Professor Harvey Sutton, in reply, said that he had come to the meeting expecting to be torn to pieces by the function supporters. But they had left him severely alone. It was interesting to note that pyorrhœa was most common in the lower incisors. If their function was unsatisfactory, then that of the upper incisor teeth must also be unsatisfactory; and pyorrhœa seldom affected the upper incisors. They could not have it both ways. The problem was a particularly interesting and difficult one, about which it was impossible to be dogmatic; but Professor Sutton thought that some of the speakers were unduly pessimistic. The incidence of dental caries was not increasing; in school children it was definitely less. The percentage of children suffering from caries had been 99%, whereas it was now below 90%. Propaganda in this field had been from 10% to 15% successful. The subject presented two problems: the building of structure and its maintenance. The hereditary theory was not convincing. Aborigines in government camps, though descended from a caries-free race, were found to be just as prone to caries as others.

In regard to pyorrhœa, Professor Sutton said that the problem would not be solved by dealing with it in its ultimate aspect; the solution could come only from beginning at the beginning.

Dr. Hansman, in reply, said that some of the confusion which had resulted from the papers was probably due to the fact that while other speakers were concerned with periodontal disease as well as dental caries, he had concerned himself with dental caries alone. They must recognize that dental caries affected only erupted teeth and that, once erupted, the enamel could no longer be changed by nutritional means, but was dissolved by an acid. Several speakers had stressed the feeling that the

modern diet was not so good as that of former times, but there was very little evidence that this was so. The modern diet was a much more mixed diet and contained sufficient calcium and phosphorus for normal development. Though the calcium and phosphorus requirements had been placed at a comparatively high figure by certain workers in America, it was known that perfect bone and teeth development could take place on intakes far below the so-called adequate minimum requirements. In the family of ten referred to, the diet contained large quantities of carbohydrate and the calcium and phosphorus content was low, yet these children had perfect physical development and good teeth.

Dr. Hansman was very interested in what Dr. Dods had said. He thought that his dilemma could be at least partially explained. Mothers might be told that their children must not have fermentable carbohydrates or sweets, but that did not mean that the children did not get them; it was well known how hard it was to restrict children in this direction. He could not agree with Dr. Beckett when he spoke of people in sedentary occupations eating so little that they had an insufficient intake of calcium and phosphorus for maintenance. People in sedentary occupations on the whole ate a good variety of food, and as Dr. Cathcart had shown in his detailed examination of the diets of people in various occupations at St. Andrews, in Scotland, the intake of food amongst those of a sedentary occupation was in general equal to that of those doing moderately hard work. He explained this partly as being due to the fact that so-called sedentary workers were often quite energetic and went in for vigorous exercise, whilst the labourer, after his day's work, was indolent. The sedentary workers apparently needed this large intake of food, because they were as a class no stouter than those following other occupations, showing that there was no luxus consumption.

With regard to Dr. Beckett's salt theory, Dr. Hansman said that there was considerable calcium content in common salt.

Vitamins must be given their proper place. They were essential for normal life. In their complete absence profound disorders of metabolism resulted, but they were no more important for life than any other essential salt, amino-acid or other necessary foodstuff. The vitamins were required only in small amounts, and though Dr. Hansman recognized their essential importance, he felt that there was a tremendous amount of humbug talked about them. There was no proof that there was a lack of vitamins in the modern diet in a country such as Australia. When they considered the increased quantities of milk and fruit consumed, it was evident that the diet was better now than it had been a hundred years ago.

The problem of dental caries must be approached with an open mind, and facts alone were of any importance. Dr. Hansman considered that the work done by the Pittsburgh and Michigan workers was of a high scientific standard and had shown ample proof that caries was simply the result of the *Bacillus acidophilus* being able to produce acid that dissolved the enamel of the tooth.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Claremont Hospital for the Insane on March 18, 1934. The proceedings of the meeting, together with the annual report of the Council, were published in this journal on July 7, 1934.

Proteosuria.

DR. GWYNNE WILLIAMS read a short paper on proteosuria. He said that about three years ago, at a Branch meeting held at the Children's Hospital, he had asked a simple question, namely: had the lecturer found the presence or absence of proteose in the urine of any assistance in differentiating rheumatoid arthritis from osteoarthritis? The reply was equally simple, but more emphatic: "I have not found the presence or absence of proteose of any assistance." Subsequently the physician had explained that he had not, up to that time, tested for proteosuria. This incident had stimulated Dr. Williams to undertake a series of simple tests. Prior to that time he had done no more than try the "ether reaction" on a number of specimens of urine which were sent to the laboratory daily for quite different investigation. This he had done largely to accustom himself to the technique.

The results, however, had soon proved interesting. The specimens were from nine epileptic patients having ketogenic diet. The tests were done twice a day for six days. Three out of nine had given positive reactions almost every time, while two gave completely negative results. Dr. Williams had failed to find any obvious clinical differences to which this could be attributed. He therefore turned again for guidance to such literature as he had. He referred to articles by Oriel and Barber: "A Study of Allergy", *The Lancet*, November 17 and 24, 1928, and "A Proteose in the Urine Excreted in Anaphylactic and Allergic Conditions", *The Lancet*, August 2, 1930.

The following was the description by Oriel and Barber of the ether test for the detection of the proteose:

> To a few cubic centimetres of urine are added first a little of a 25% solution of sulphuric acid and then ether to form a layer above the urine about half an inch in depth. The mixture is then thoroughly shaken with the thumb over the mouth of the test tube and allowed to stand. The layer of ether becomes frothy, but if the reaction be positive the froth in its lower part becomes viscous and opaque, like melted wax after ten minutes or so. In strongly positive tests the tube of urine can then be inverted without spilling. The degree of positiveness is measured by the depth and density of the waxy scum beneath the superficial froth, and we should here emphasize that the test is one of degree only, because a slight reaction—*i.e.*, the formation of a narrow band of opaque scum—is frequently found in apparently normal people. For this reason we do not wish to exaggerate the importance of the reaction; nevertheless it is one of great interest and deserves further study.

For his part, Dr. Williams had disregarded the narrow opaque band between the two fluids, calling the results of those tests negative. Only once had he been able to invert the tube, owing to the marked viscosity of the ethereal layer.

This was no occasion to discuss the chemistry of the substance shown by this reaction; it would suffice to say that it was a proteose (hereinafter called the proteose) and that it could be precipitated from ether by alcohol. Moreover, it could be redissolved and prepared for injection if required.

The conditions in which Oriel and Barber found the proteose were prurigo, urticaria, hay fever, asthma *et cetera*, and migraine, and owing to the narrow borderline between some forms of epilepsy and migraine, the hope naturally arose of the possibility of finding an important allergic factor in some epileptics and of subsequently devising some appropriate treatment. Instead of rushing to try intradermal reactions and possibly being sidetracked (there had been severe criticism of these), Dr. Williams had determined to continue with the ether test until he had over a thousand tests upon which to base his further actions. This appeared to have been a wise course in view of the findings.

The three patients who had given such persistently positive results in the early series had in the larger series continued to give mostly positive results. Number 1 had given completely negative results 26 times out of 73 tests. Number 2 had given completely negative results six times out of 65 tests. Number 9 had given completely negative results only four times out of 72 tests.

Dr. Williams had failed to find any apparent coordination between the presence of proteosuria and the occurrence of epileptic fits. At first these appeared in two or three of the cases to be a cycle of increasing proteose followed by a fit and then a negative phase. Indeed, he had tried his hand at forecasting on this basis when fits would occur. After a few early successes he lapsed and enjoyed no more luck than the local meteorologist.

A disquieting factor was that routine specimens which arrived in the laboratory in the ordinary way frequently showed positive reactions of marked type. This seemed

to be particularly so in pyrexial conditions—pneumonia, typhoid fever, malaria *et cetera*. In two cases of serum sickness the proteose reaction was positive.

Controls were then carried out with five healthy males (members of the office staff) and members of the female nursing staff. Examination of these results seemed to show that a moderately positive ether reaction was of little, if any, significance.

With the provision that the persistence of the reaction in three epileptic cases, 1, 2 and 9, was worth further investigation, Dr. Williams felt that he was now of the same opinion as the physician who had so happily started him off on the work.

NOMINATIONS AND ELECTIONS.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Myers, William Keith, M.B., B.S., 1933 (Univ. Sydney), 5, Selwyn Road, Wollstonecraft.

Theile, Egmont Friedrich Hellmuth, M.B., 1933 (Univ. Sydney), Sydney Hospital, Sydney.

The undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Cohen, Cecil Ashley, M.B., B.S., 1931 (Univ. Melbourne), 31, Mitford Street, St. Kilda, S.2.

Correspondence.

HYPOGLYCÆMIA AND KETOSIS: THEIR RELATIONSHIP TO CHRONIC ANTRAL DISEASE AND BRONCHIECTASIS.

Sir: Dr. Brown's letter in the journal of September 22, 1934, criticizing my paper, is surely redolent of indignation that the physician should attempt to encroach upon the sacred field of rhinology.

Dr. Brown states that hypoglycæmia probably exists in a certain number of the ordinary population, and for this reason considers that my conception of hypoglycæmia and consequently that of many observers in all parts of the world is based on a false standard. Harris, in America, found that 4% of 1,861 cases examined had a subnormal blood sugar. Arguing on a similar false logic to Dr. Brown, it could be maintained that because a very large percentage of the population have anatomical nasal abnormalities, then these are of no pathological significance. This, of course, would be absurd.

I have never denied the probability of some truth in his own theory of restricted airway, but there must be some other factor than such anatomical abnormality to produce sinus disease with bronchiectasis, otherwise this distressing state would be even more common than at present. Dr. Brown's views concerning antral disease and bronchiectasis have been well known to me for some time and, like him, I was "not thoroughly satisfied with the present line of treatment". Previously, treatment of anatomical abnormalities seemed to me to be rational, but the disappointing results often seen stimulated me to search for some other factor. I have suggested a metabolic disturbance as a possible common ætiological factor of both sinus and chest changes. If Dr. Brown will refer to my original article he will find the following statement: "Evidence has been produced in support of the view that hypoglycæmia and ketosis are important pointers to the underlying ætiology of at least some cases of antral disease and lung fibrosis."

The case which Dr. Brown described actually supports my theory of some metabolic disturbance, as he has shown that a change in the diet will cause a disappearance of symptoms and an apparent "cure".

Dr. Brown has stated, when dealing with catarrhal illnesses in English schools: "These facts suggest that it may be the sugar that is responsible for these troubles, which is the opposite of Dr. Sippe's theory." Apparently my letter, as well as my original article, has again been misread, as I particularly pointed out that hypoglycæmia was not in itself the cause of the condition under discussion, but merely one sign of some general metabolic upset.

Dr. Brown states that my previous article concerning hypoglycæmic angina was criticized by Dr. Duhig. He may be interested to know that Middleton and Oatway, and Shirley Smith in England, and Willgenstein and Mendel, von Hagnal and Schaffer, de Lauteen and Baumann, Weichmann and Kock, and Stepp and Parade on the Continent, and Young in America have propounded the same findings, although all these results were not known to me when I first reported the electrocardiographic changes.

Apparently, when speaking of glucose and mental disorders, Dr. Brown referred to the work of Quastel and Strom-Olsen on the use of glucose and insulin in the prolonged narcosis treatment of psychiatric conditions. This, of course, has no bearing whatever on the subject of hypoglycæmia, as there was no suggestion of a lowered blood sugar in these cases. The aim of glucose and insulin therapy was to provide adequate nutrition for vital organs during the diminished metabolism associated with narcosis.

With regard to his passage concerning "catarrhal" diseases in English schools, he refers doubtless to an article by J. H. Paton in the *Edinburgh Medical Journal*, a criticism of which I had in my original article, but deleted in order to shorten the paper. The observations contained therein would seem to any thinking observer to be too cursory, as sugar and carbohydrates are the only articles of diet mentioned. It is very well known that during the war period butter was completely eliminated from these diets. It may with equal validity be stated that the absence of butter was the cause of the lessened incidence of catarrhal illness—possibly this may even be nearer the truth, on account of the tendency for butter to produce acidosis.

On page 678 of my original paper I suggested that "this condition is very closely linked to the allergic state". "Allergy" is admittedly not an entirely satisfactory term, but serves to describe a well recognized group of individuals who are extraordinarily sensitive to either ingested or inhaled antigens and who tend to develop antral disease and bronchiectasis. Lapage has aptly described this group as possessing "an exudative diathesis". Diathesis or type is rightly becoming more and more recognized as having a very important bearing on the development of various morbid states.

Yours, etc.,

Clive Sippe.

Brisbane,
October 1, 1934.

THE PAN-PACIFIC SURGICAL ASSOCIATION.

The following letter has been forwarded for publication by Sir James Barrett, Melbourne.

Sir James Barrett,
105, Collins Street,
Melbourne.

My dear Sir James: You will, no doubt, receive a letter shortly from Dr. Pinkerton, Secretary of the Pan-Pacific Surgical Association, making inquiries relative to the proposed meeting to be held in San Francisco in 1935. The plan is to hold this meeting prior to the meeting of the British Medical Association in Australia so that we might catch some of the men going to that meeting. It would, therefore, make it possible for those of you who come over to return with the British delegates. We are quite anxious to have a large representation from Australia and New Zealand. We are mindful of the fact, of course, that there has been a severe depression and that times are exceedingly hard. But we thought we could get a few

delegates from each and have the meting in San Francisco next year and the following meeting in 1937 in Australia or New Zealand.

I am wondering if you will contact some of the men in your vicinity who will be willing to furnish papers and attend the Conference in San Francisco, and let us know what can be done about it. We are all looking forward to a reunion of the friendships made at the first meeting. We hope that the depression will be over by that time and that things will look much brighter.

Sincerely yours,

George W. Swift, M.D.,
President.

September 13, 1934.

REMOVAL OF FOREIGN BODIES FROM THE EXTERNAL AUDITORY MEATUS.

Sir: A recent article on treatment of ear conditions explained how to remove a tight foreign body from the meatus by coaxing past it a rod with a right-angled bend at the tip. This needs a general anæsthetic.

Years ago I read a much better tip in *The British Medical Journal*. I have used it only once, when it acted like a charm.

Dry the meatus. Take a quill iodine brush, with secure bristles well rubbed in dental cement, and pass it into the meatus till the cement is in contact with the foreign body. No anæsthetic of any sort is needed. After ten minutes' wait, withdraw everything.

Yours, etc.,

J. B. Hogg, M.B.

Deepwater,
New South Wales,
October 4, 1934.

THE CONSERVATIVE TREATMENT OF FACIAL PARALYSIS.

Sir: To Dr. Royle's instructive survey may I add the suggestion of employing diathermy for catarrhal cases. It has a good effect even upon late cases. In early cases it acts like a charm.

Yours, etc.,

W. Kent Hughes.

22, Collins Street,
Melbourne,
Undated.

INTUSSUSCEPTION.

Sir: Dr. Hipsley's remarks (The Medical Journal of Australia, October 6, 1934) about the effect of a long mesocolon in maintaining the blood supply in intussusception prompts me to report the following case.

A male infant, aged seven months, and previously healthy, had been ill with digestive upset and loose green motions for about a week. He was then seized with severe abdominal pain and vomiting and was given castor oil and an enema by his mother without relief. The pain persisted with brief intervals for nearly sixty hours, no fæces being passed, but only blood-stained mucus. Vomiting became severe and the child seemed to his mother to be getting worse. Seen at this stage, he was found to be dehydrated and collapsed, with a subnormal temperature. There was a large mass in the left side of the abdomen, below the umbilical level and extending down into the pelvis. The right side was empty, and on rectal examination a soft elongated tumour was felt almost at the anus.

At operation later there was a good deal of turbid fluid in the peritoneal cavity. The intussusception was reduced completely with a little difficulty. The bowel was wholly viable, but the caecal wall was congested and œdematous, and a nodule in this region seemed to be the apex. The bowels acted naturally within twelve hours and convalescence was uneventful, except that in the third week the infant developed a diffuse fusiform enlargement of the first and second phalanges of the middle two fingers of the right hand. This subsided to a large extent, but left some thickening.

Interest is lent by the mother's statement that a brother of the patient had died at the same age with a similar condition after an illness of three weeks.

Yours, etc.,

Frederick Kelleher, M.B., B.S.

Forbes,
New South Wales,
October 6, 1934.

THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN.

Sir: An intimation was previously made in your journal that the board of management of this hospital had decided to permit medical practitioners generally to have non-infectious medical cases admitted to the private and intermediate divisions under their care.

I now have pleasure in informing you that the board has decided to extend a similar privilege in regard to the admission of infectious cases (other than diphtheria) to the private and intermediate wards of the Charles Clubbe Memorial Isolation Block.

Yours, etc.,

S. W. G. Ratcliff,
Chief Executive Officer and
Medical Superintendent.

Sydney,
New South Wales,
October 12, 1934.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A meeting of the Senate of the University of Sydney was held on October 8, 1934.

The Diploma in Tropical Hygiene was awarded to Bruce Robson Overend, M.B., Ch.M.

The Senate accepted with grateful thanks an offer from Professor C. E. Fawsitt of the sum of £100 for the purpose of providing in 1935 two prizes in chemistry, to be awarded to deserving students in that subject.

In view of the approaching visit to Sydney of His Royal Highness the Duke of Gloucester, the Vice-Chancellor was empowered to make any alterations considered requisite in the time-table of the annual examinations, scheduled to commence on November 19.

Dr. Alwyn James Arnott was appointed by the Senate of the University to succeed Professor R. Fairfax Reading in the Chair of Dentistry. The new Professor, who is to take up his duties on January 1, 1935, was born in December, 1899. He graduated Bachelor of Dental Surgery in Sydney in 1921, and was admitted to the degree of Doctor of Dental Science in 1928. He is a Fellow of the International College of Dentists, as well as a Fellow of the American College of Dentists. He is at present Superintendent of the United Dental Hospital of Sydney and Lecturer in Oral Surgery in the Faculty of Dentistry.

Proceedings of the Australian Medical Boards.

VICTORIA.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as duly qualified medical practitioners:

Carroll, Arthur James, M.B., B.S., 1934 (Univ. Melbourne), Saint Vincent's Hospital, Melbourne, N.6.

Ginsburg-Lapin, Mollie Amelia, M.B., B.S., 1930 (Univ. Sydney), Walpeup, Victoria.
Grainger, Francis Transvaal, M.B., Ch.M., 1925 (Univ. Sydney), Grenville Street South, Ballarat.
Matthews, Ian Hamilton Killough, M.B., B.S., 1934 (Univ. Melbourne), 91, Tooronga Road, East Malvern, S.E.5.
McPherson, John, M.B., Ch.B., 1909, M.D., 1918 (Aberdeen), 33, Bay Road, Sandringham, S.8.
Peters, Shiela Rosa, M.B., B.S., 1933 (Univ. Melbourne), 39, Waverley Road, Caulfield, S.E.8.

Additional diplomas registered:
Lowe, Thomas E., M.D. (Melbourne), 1934.
Mancy, Ernest H., M.D. (Melbourne), 1933.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 *to* 1933, as duly qualified medical practitioners:

Clouston, Thomas Bennett, M.B., 1905 (Univ. Sydney), Mackay.
Shiels, Douglas Oswald, M.B., B.S., 1933 (Univ. Melbourne), Mount Isa.

Additional qualification:
Johnson, Horace William, M.R.C.P. (Edinburgh), 1934.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, as a duly qualified medical practitioner:

Maguire, Frederick Arthur, M.B., 1911, Ch.M., 1919, M.D., 1926 (Univ. Sydney), F.R.C.S., 1919 (England), New South Wales Medical Board, Macquarie Street, Sydney.

Obituary.

FREDERICK CHALLANDS.

WE regret to announce the death of Dr. Frederick Challands, which occurred on September 13, 1934, at Brisbane, Queensland.

ERNEST THORBURN MACGOWAN.

WE regret to announce the death of Dr. Ernest Thorburn Macgowan, which occurred on September 26, 1934, at Hobart, Tasmania.

DANIEL KELLY.

WE regret to announce the death of Dr. Daniel Kelly, which occurred on October 14, 1934, at Sydney, New South Wales.

NOTICES.

WE have been asked by the Honorary Secretary of the Fourth Division, Army Medical Corps Association, to make the following announcement:

> The annual reunion of the Fourth Division, Army Medical Corps Association (Fourth, Twelfth and Thirteenth Field Ambulances, Australian Imperial Force) will be held at The Palms, Alexandra Avenue, Melbourne, on November 12, 1934. The officers of the Association are making special efforts to insure a large attendance of members and visitors attracted from other States by the Melbourne Centenary celebrations. An appeal is made to former medical officers of the units mentioned to attend. The Honorary Secretary's address is: 16, Wolseley Street, Mont Albert, E.10, Victoria.

THE Orient Line has made available to the Royal Prince Alfred Hospital, Saint Vincent's Hospital and the Royal North Shore Hospital of Sydney the Royal Mail Steamer *Orontes* for a ball on the evening of November 20, 1934. The full proceeds of the ball will go to the hospitals.

The ball is under the patronage of Their Excellencies the Governor-General and Lady Isaacs, and His Excellency the Governor of New South Wales and Lady Game.

The liner will lie at Number 7 Wharf, Woolloomooloo Bay. Tickets are one guinea each. The ticket secretaries are: Royal Prince Alfred Hospital, Mr. F. W. Wilson; Saint Vincent's Hospital, Mrs. P. L. O'Halloran; Royal North Shore Hospital of Sydney, Miss Jean Brewster. Tickets may also be obtained at the office of the Orient Company, 2, Spring Street, Sydney, or from W. H. Paling and Company, George Street, Sydney.

Corrigenda.

Two errors have occurred in the Jackson Lecture, by Dr. Eric Jeffrey, published in the issue of October 6, 1934. On page 442, in the last line of the first column, "three years" should read "thirteen years"; and on page 445, in the first column, line thirty-nine, "rhetorician" should read "rhetoricians' ".

IN the abstract of a paper by A. Rugg-Gunn, published in the issue of October 13, 1934, Hertzian waves with a wave-length of 10^{-7} to 10^{-13} Angström units are mentioned. The figures should read "10^{7} to 10^{13}".

Books Received.

THE CIRCULATION OF THE BLOOD, by W. Parsons, M.A.; 1934. London: The Sheldon Press. Crown 8vo., pp. 204, with illustrations. Price: 5s. net.

A STUDENT'S HANDBOOK OF CLINICAL ELECTROCARDIOGRAPHY, by W. Evans, M.D., M.R.C.P.; 1934. London: H. K. Lewis and Company, Limited. Medium 8vo., pp. 60, with illustrations. Price: 5s. net.

BIRTH CONTROL TO-DAY: A PRACTICAL HANDBOOK FOR THOSE WHO WANT TO BE THEIR OWN MASTERS IN THIS VITAL MATTER, by M. C. Stopes; 1934. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 237, with illustrations. Price: 5s. net.

STAND UP AND SLIM DOWN: BEING RESTORATION EXERCISES FOR WOMEN, WITH CHAPTER ON FOOD SELECTION IN CONSTIPATION AND OBESITY, by E. A. Hornibrook; Sixth Edition; 1934. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 181, with illustrations. Price: 6s. net.

THE CONSTITUTION AND ITS REACTION IN HEALTH, by T. E. Hammond, F.R.C.S.; 1934. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 169. Price: 7s. 6d. net.

EXPERIMENTAL PHYSIOLOGY, by E. Sharpey Schafer, F.R.S.; Fifth Edition; 1934. London: Longmans, Green and Company. Demy 8vo., pp. 175, with illustrations. Price: 6s. net.

DISEASES OF WOMEN, by Ten Teachers, under the direction of Comyns Berkeley, M.A., M.D., M.C., F.R.C.P., F.R.C.S., M.M.S.A., F.C.O.G.; Fifth Edition; 1934. London: Edward Arnold and Company. Medium 8vo., pp. 580, with illustrations. Price: 18s. net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, edited by E. B. Krumbhaar, M.D.; XIV: Medicine in Persia, by C. Elgood, M.D., M.R.C.P.; 1934. New York: Paul B. Hoeber. Foolscap 8vo., pp. 118, with illustrations. Price: $1.50 net.

Diary for the Month.

OCT. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 24.—Victorian Branch, B.M.A.: Council.
OCT. 25.—New South Wales Branch, B.M.A.: Branch.
OCT. 25.—South Australian Branch, B.M.A.: Branch.
OCT. 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. B. Williams (B.M.A.) has been appointed Medical Officer of Health by the Mount Magent Road Board, Western Australia.

* * *

Dr. P. W. Shanahan (B.M.A.) has been appointed Medical Officer of Health by the Meckering District Road Board, Western Australia.

* * *

Dr. R. N. Reilly has been appointed Honorary Anæsthetist at the Adelaide Hospital, South Australia.

* * *

Dr. L. V. Darby (B.M.A.) has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be a Certifying Medical Practitioner at Inglewood, Victoria.

* * *

Dr. R. Nettleton has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be a Certifying Medical Practitioner at Orbost, Victoria.

* * *

Dr. A. L. Kenny (B.M.A.) has been appointed, pursuant to the provisions of Section 74 of the *Lunacy Act*, 1928, to be an Official Visitor to visit the metropolitan mental hospitals within twenty miles of the General Post Office at Melbourne, Victoria.

* * *

Dr. J. Smith-Guthridge has been appointed Government Medical Officer, Tea Gardens, Victoria.

* * *

Dr. T. Godlee (B.M.A.) has been appointed Medical Officer of Health by the Northampton Road Board, Western Australia.

* * *

Dr. S. P. Barnett and Dr. F. F. Pincus (B.M.A.) have been appointed, pursuant to the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Act of* 1933, Queensland, to be Members of the Medical Board constituted by that Act.

* * *

Dr. F. H. Wilson (B.M.A.) has been appointed Senior Medical Officer, Office of the Director-General of Public Health, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi, xvii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Radio-Diagnostician.
CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Medical Officers.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
GEELONG AND DISTRICT HOSPITAL, GEELONG, VICTORIA: Senior Resident Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Assistant Ophthalmic Surgeon.
STATE PUBLIC SERVICE, QUEENSLAND: Medical Officer.
THE UNIVERSITY OF SYDNEY, NEW SOUTH WALES: Pathologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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